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MALIGNANT MELANOMAS

With Report of 4 and 7 Year Cures

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MALIGNANT melanomas are recognized to be rapidly fatal tumors, and although their number is low in proportion to other malignancies, their occurrence is fairly common. Treatment has often been considered hopeless and therefore a decision as to choice of treatment has been confusing.

Pathologists have referred to the growths both as sarcomas and carcinomas, thus adding to the uncertainty of their origin. The majority of these lesions first appear on the surface of the body where pigment is common, but primary growths have been described in unusual sites, and according to one theory can originate in any ectodermal tissue. They may arise in the skin, the nail bed, the choroid of the eye, the mucous membrane of the mouth, rectum, or vagina, and have been found in the meninges and parotid gland.

Destruction of the precancerous lesion is of prime importance, and since a large percentage of such malignancies arise in pigmented nevi, if these simple looking growths could be removed adequately at the slightest suspicion of growth or activity, many patients might be spared (Fig. 11).

The question of removing moles, either for appearance or for the possibility of cancer is often put to the doctor, and therefore some rules are advisable for determining which moles had best be removed. Nearly everyone has pigmented moles in profusion, but it would be impossible to remove all of them. The safest procedure is to destroy all moles that are subjected to chronic irritation, as in shaving, friction of clothing, combing the hair, fingering because of habit, repeated pulling of hairs, etc., whether they are pigmented or not. Every lesion should be removed that shows evidence of growth, increase in vascularity, change of color, repeated infection or ulceration. The smooth, coal-black nevi should likewise be removed.

In the congenital growth that is apt to be flat and not heavily pigmented, the basement membrane may be lacking and the cells may be scattered all around as they appear in a malignancy, and yet this congenital type rarely actually becomes malignant and might even be considered as an anomaly of the skin rather than a tumor. Dr. N. A. Womack classifies this type of growth as neuro-nevi. On the other hand, the developmental mole that appears at any time during life and progresses in size is probably the type that most often becomes malignant, and this point of whether a growth is congenital or developmental may be of some importance in deciding about removal.

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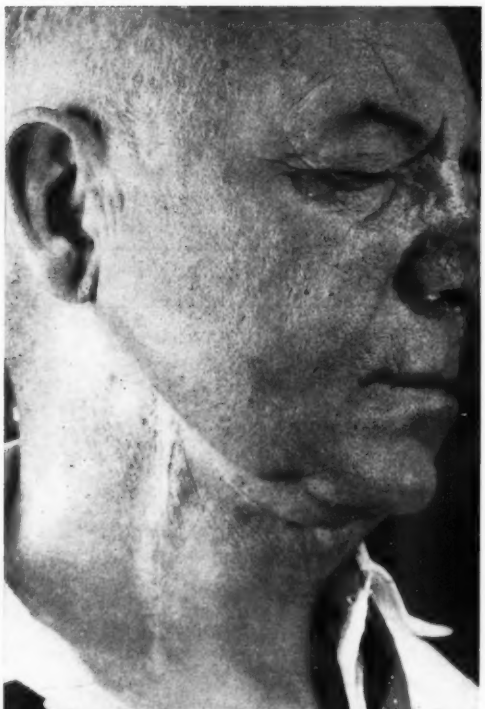


Fig. 1. a, Scar of radical neck dissection for removal of metastatic melanocarcinoma from the temple where a healed scar is also shown. Patient well 7 years.

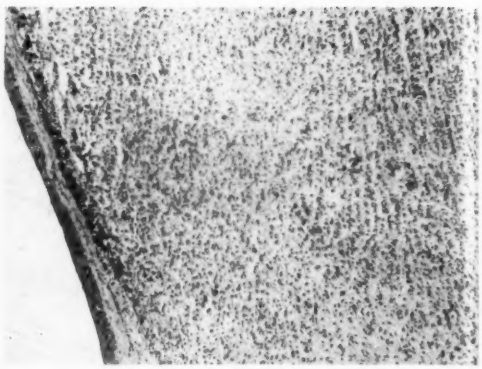


Fig. 1. b, Photomicrograph of gland showing complete replacement with melanocarcinoma.

Since there have been instances of activation and the development of cancer by removing or "fooling" with a common mole, many patients, and doctors as well, are afraid of any form of treatment and recommend that all moles be left alone. Unquestionably, some practices of therapy are dangerous, such as pastes and acids, and even electrodesiccation when done without anesthesia, in which circumstance the inability of the patient to cooperate hurries the operator. It has been noted by others, as well as ourselves, that multiple electrodesiccations have been done in many of the terminally fatal lesions.

The removal should be quick, painless, nonirritating, thorough, and should involve the minimum handling of the growth. In quiescent lesions, clean knife excision, carried out in such a manner is acceptable. Our usual method, however, in small lesions is to use novocain without inserting the needle in the growth, and then completely and thoroughly to destroy the area with a small pointed electric cautery, which is made to penetrate the entire depth of the skin and enter the underlying fat. A circular burn is thus produced that requires dressing for 1 to 3 weeks. The ultimate scar is smaller than the area of destruction and after 6 months the area has usually whitened and smoothed out so that the patient has little objection to the scar (Fig. 11). In a few instances of keloid or heavy scar formation, a repair is made later. For larger lesions, this simple, quick cautery excision may also be done, but patient should understand repair will be necessary later.

It has been stated that melanomas arising from non-pigmented areas are the most malignant, but so far all we have seen have been pigmented.

DIAGNOSIS

The change from an innocent pigmented nevus to a malignancy should not be difficult to detect and diagnose. At its onset there may be an increase in elevation or of surface area, or the skin may retain its usual texture with a deepening of pigmentation to a velvety black. Other early signs are an increase in vascularity and an apparent chronic infection with slight tenderness. The appearance of an

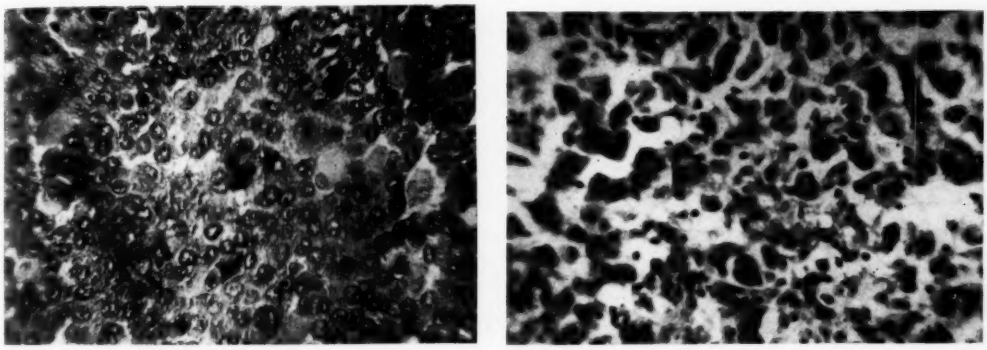


Fig. 2. a, left, Photomicrograph of primary tumor in parotid gland. b, Skin metastases from melanocarcinoma of parotid.

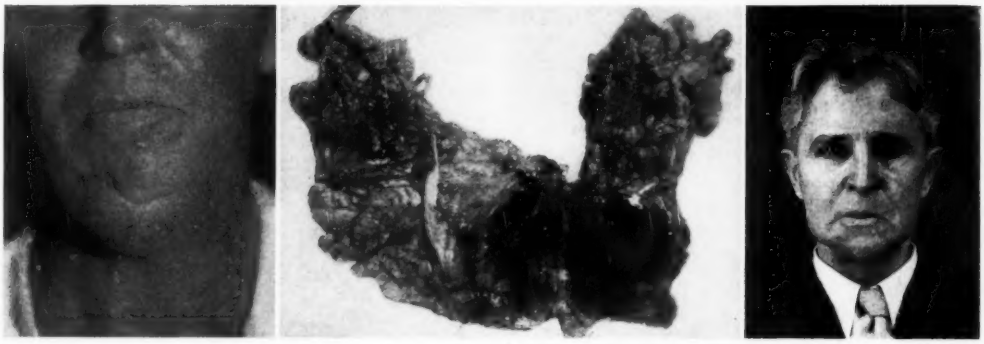
entirely new growth may be difficult to diagnose as a melanoma; but a localized process in the skin or under the nail presenting an increase in bulk and lacking the pain and tenderness of an infection and the characteristics of a wart should at least be considered as a malignant neoplasm. Obvious melanin may not be present; one such lesion on the heel in our series had two small flecks upon its surface which could have been taken for tiny blood clots (Fig. 9).

TREATMENT

Malignant melanomas of the face and head. The local lesion. Death almost never occurs from extension or erosion of the local lesion, as may be the case in other malignancies of the face. This is probably due to the fact that hopeless metastases and death occur so rapidly.

Treatment is limited to surgery since there is almost complete agreement that melanomas are resistant to radiation. This does not mean that most any method of local eradication may suffice or be of equal safety, and it is certain that tampering with the local lesion by anything less than complete wide removal is merely inviting trouble. The surgical procedure should be quick, should avoid all irritation of the lesion, should require the minimum handling of the field, and should remove a generous margin of normal surrounding skin. In addition, the un-

Fig. 3. a, left, Healed scar of removal of black mole from lobe of ear and metastasis below jaw 2½ years later. b, Scar of thorough but limited dissection. Patient died accidentally 1½ years later without recurrence.



a b c

Fig. 4. a, Healed primary site on chin after acid and paste treatment but with huge neck metastasis. b, Neck mass removed. c, After operation. Patient died in 1½ years from huge metastases above clavicles.

derlying subcutaneous tissue down to the fascia should be removed; usually a greater area of this than of skin, is taken and it may be necessary to sacrifice a facial nerve or the full thickness of the cheek. It is usual to employ the cautery or diathermy in the removal. Repair of the resultant defect may require only a free skin graft, but some restorations, of course, may involve the use of a pedicle flap.

Metastases from melanomas in general. Such growths require the earliest possible widespread removal to avoid metastasis; as to the time element in metastasis, one might almost compare hours in melanomas to weeks in other carcinomas following treatment of the local lesion. Five possibilities are present:

1. The initial lesion may recur following removal or remain cured (Fig. 1).

2. Metastases may occur in the adjacent skin in single or multiple patches of melanin. These may vary from uniformly deeply pigmented velvety areas to stippled smutty patches. This is probably a precursor of widespread metastases.

3. The most hopelessly swift spread of all is the sudden appearance of skin metastases in many parts of the body, with or without evidence of visceral involvement. This widespread distribution has raised the question of whether this is a blood borne metastasis or the sudden activation of melanin producing cells normally present throughout the body.

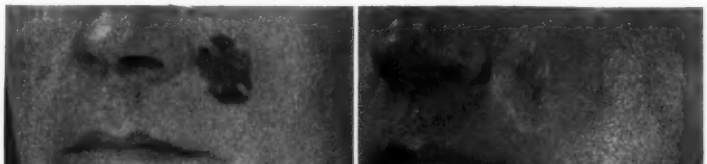


Fig. 5. a, left, Slowly growing mole of 15 years' duration with new activity in lower part. b, Scar of removal and grafting primary area and neck dissection. Gross involvement found in neck. Patient well 15 months to date, having had a metastasis removed from the body of the parotid gland in the meantime.

Fig. 6. Primary melanocarcinoma of the parotid with paralysis of the seventh nerve. Death some months after radical operation.

Fig. 7. Primary scalp melanocarcinoma developing in an old mole. No regional gland involvement but death with stomach metastases.

Because of the relationship between the pituitary secretion and the melanophore system, Wigby and Metz[1] treated such a patient in June, 1938, with x-ray therapy to the pituitary gland. At the date of their publication in February, 1939, there had been complete retrogression and recovery of the patient. Patients treated subsequently, which were not of this type but fell in the group of local gland involvement did not respond (Fig. 8).

4. The local lesion may remain cured but later, from a few months to 25 years, the patient may die of a visceral metastasis. Most of the patients with a long period of alleviation seem to have had the original lesion in the choroid of the eye. This must be a blood stream spread, but the malignant cells may have been resting in the glands.

5. The last group and the most important, because it is in these that some hope of cure may be held, contains those in which the primary focus is eradicated and the metastasis appears in the regional lymph nodes, there to be held in check for some indefinite period. Radical block dissection of the neck has shown definite promise and gives patient his only chance for life (Figs. 1, 3, 4, 5).

[1]Am. J. Roentgenol., 1939, 41: 415.

Some patients may show all methods of metastases: local skin, glands, distant skin, and visceral involvement through the blood stream.

Fig. 8. Multiple skin metastases from irritated mole on forehead. Also had lymphatic involvement and blood stream metastasis to the lungs.

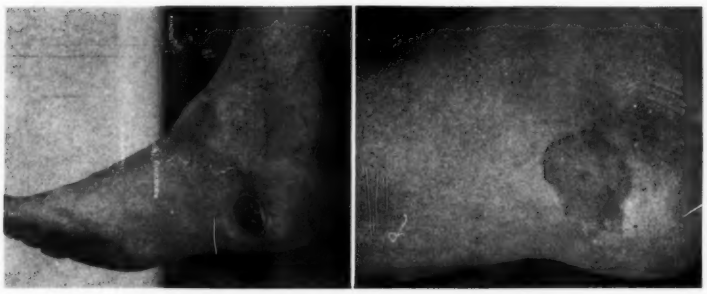


Fig. 9. Melanocarcinoma with ulceration of 6 months' duration. b, right, Result of wide removal and grafting. Inguinal involvement was removed with no recurrence, but death from abdominal metastasis resulted in 15 months.

A problem in treatment brought up by a consideration of these methods of spread concerns whether or not prophylactic regional gland dissections should be done. Against prophylactic neck dissection is the inability to tell which growths will metastasize only to the lymph nodes. However, it is well known that a gland dissection done before gross involvement occurs gives the patient a much better chance than one done after involvement is demonstrable clinically. Therefore, if the primary lesion is located so that its lymph drainage is fairly well predictable, a prophylactic dissection should be done (Fig. 4).

REPORTS OF ILLUSTRATIVE PATIENTS

The following brief case reports are included to show a few worthwhile results of endeavor and to recall some vagaries of the lesion.

CASE 1. Seven year cure following radical neck dissection for metastatic melanocarcinoma. The patient shown in Figure 1a had a mole removed (elsewhere) from the right temple; this recurred in 2 years and was removed again. Three years later a lump appeared under the angle of the jaw. When seen at that time, biopsy showed metastatic melanocarcinoma and an extremely radical neck dissection was done. Within 2 months there were many metastatic areas along the upper incision; area was widely excised. Patient has remained well 7 years with no evidence of local or distant recurrence.

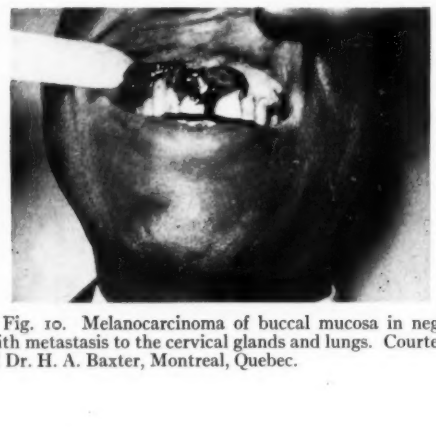


Fig. 10. Melanocarcinoma of buccal mucosa in negro with metastasis to the cervical glands and lungs. Courtesy of Dr. H. A. Baxter, Montreal, Quebec.

CASE 2. Patient with primary melanoma of parotid and secondary skin metastases, alive and well 4 years after radical removal. A 45 year old woman had a parotid tumor removed elsewhere and 6 months later there was a recurrence in the gland with subsequent appearance of a black area behind the ear. When seen 6 months later a complete removal of the tumor was done without sacrificing the seventh nerve, plus removal of the involved skin. This patient is alive 4 years without evidence of recurrence but should have a complete neck dissection done. The seventh nerve was saved because it was not felt that there was enough invasion to necessitate its removal.

CASE 3. Patient well 1½ years following removal of metastatic melanocarcinoma. The patient shown in Figure 3a shows a mass in the neck and the scar of removal (done elsewhere) of a black mole from the lobe of the ear 2½ years previously. A thorough but somewhat limited dissection of the area was done and he lived 1½ years with no sign of recurrence, when he died accidentally. He should have had a complete neck dissection done.

CASE 4. Mole on chin removed with acid and cancer paste: neck metastasis. Figure 4a shows healed primary area on chin with large cervical metastasis present 6 months. The dissected mass and the later appearance is seen in Figure 4b and c. Patient died 1½ years after operation from metastases in lower neck which was not dissected because of refusal by patient.

CASE 5. Melanocarcinoma in a mole of the cheek; neck metastasis. Figure 5a shows mole in a man of 42 years that had developed 15 years before. It

a b c

Fig. 11. a, Illustration of an ugly, slowly growing mole of the lip. b, The site of the mole after cautery removal. c, The final healing result with the practically invisible scar.

had grown and become blacker in the past 2 months, and one node under the jaw was enlarged. Figure 5b shows scar of removal and the grafting of the original area after radical neck dissection when gross involvement was found. One year after neck dissection a mass appeared in the parotid gland of the same side. This was removed, but the seventh nerve was saved. The growth was found to be melanomatous. No other recurrences 15 months later.

CASE 6. Primary parotid melanocarcinoma with seventh nerve paralysis. The patient shown in Figure 6 gave some difficulty in diagnosis because the growth was thought to be a malignant mixed tumor of the parotid. At operation, when it was found to be coal-black, it was thought at first to be from coal-tar, as he was known to have constantly chewed tar on this side of the mouth. Extremely radical operation was done but death resulted in a few months, presumably from distant metastases.

CASE 7. Melanocarcinoma of the scalp without gland involvement but with death from metastasis to the stomach. Figure 7 shows the primary scalp growth that was irritated in combing the hair. It was treated (elsewhere) with the "electric needle" and lanced twice. The area was widely excised and grafted. Four months later death occurred and he was found to have melanocarcinoma around the pylorus. No local recurrence or cervical gland involvement was present.

CASE 8. Melanocarcinoma with skin, gland, and pulmonary metastases. Figure 8 shows, rather poorly, the multiple metastases following chemical irritation and growth of a mole in the temple. A problem of some academic interest even though it is of little logic, was involved in the treatment carried out in this patient, namely; since this patient was slowly developing melanotic areas over his whole body, it was conjectured that vitiligo might be an opposite condition. Therefore as a negro was present who had become practically white over a period of years, some of his blood and parts of his skin were put under the skin of this patient. The disease progressed, however, and pulmonary death occurred 1 year later.

CASE 9. Melanocarcinoma of foot, local and inguinal region control of growth, with death from abdominal metastasis. The patient in Figure 9 is included in this group as a matter of interest. The rather small original lesion was controlled by removal and grafting; inguinal involvement was removed with no recurrence following, but the patient died after 16 months with abdominal metastasis. This growth had developed in an occupational callus which had ulcerated and became granulomatous in appearance over a period of 6 months.

CASE 10. Melanocarcinoma in arm, removed and grafted. Death 4¾ years later with metastasis in brain but with no local recurrence. A melanocarcinoma occurred in a developmental mole of 7 years' duration in a woman of 36. Wide excision and grafting was done. Nearly 5 years later she developed a metastatic gland above the opposite clavicle and soon died with a cerebral involvement. There never was any local recurrence or evidence of axillary metastases on the same side.

CASE 11. Occurrence of melanocarcinoma and adenocarcinoma of sigmoid in one patient. A 40 year old man had a reddened area on his leg 11 years previously. Four years later it was traumatized and ulcerated. Three years later a melanocarcinoma was evident and it was removed. Four years later the local area was well but melanocarcinoma was dissected from the inguinal region. Four months after this there was intestinal obstruction necessitating a colostomy, and another malignancy was found to be present, an adenocarcinoma of the sigmoid. Death occurred 3 months later with no evidence of the melanocarcinoma.

THE PATHOLOGICAL PHYSIOLOGY OF JOINTS

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As early as 1742, William Hunter discussed the structure and diseases of the articulating cartilage; in 1794 Edward Ford set down his observations on joint affections; Percivall Pott's teachings were compiled in 1808; Brodie in 1834, and Barwell in 1881 devoted comprehensive volumes to the pathology and surgery of joint diseases; and in 1879 Henry Morris wrote an excellent book on the anatomy of the human joints. In recent years, Nichols and Richardson, Leriche and Policard, Knaggs, Phemister, Fisher, Sigurdson, Pemberton, Key, Kling, and many others have made definite contributions. There are as yet numerous unsolved problems concerning the diseases of the joint. But there is a great deal known and the persistent disregard as to what makes up a joint has clouded our understanding of what goes on within it, in health or disease.

THE ORIGIN AND CLASSIFICATION OF JOINTS

All the intrinsic elements of the joint, except the nerve fibers and the nucleus pulposus of the intervertebral fibrocartilage, arise from mesoderm. In the region of the future skeleton, the mesoderm undergoes circumscribed condensations which are later transformed into cartilage and bone. The joints are formed from the embryonic connective tissue binding these segments. The fate of this interposed tissue determines the type of joint that is developed later.

In synarthroses the embryonic tissue uniting the skeletal anlage remains a continuous substance though it may differentiate into a thin membrane, a ligament, or a cartilaginous plate. Respectively, the joint is called sutura, syndesmosis, and synchondrosis. The union between the bodies of the vertebræ presents a modified form of synchrondrosis in that the intervening tissue is fibrocartilage and contains the remnant of notochord, nucleus pulposus, which provides the joint with a springy, resilient motion. For this reason, the articulation between the bodies of vertebræ is called amphiarthrosis (12), meaning a joint with slight motion, as synarthrosis represents joints with no motion and diarthrosis is the term applied to joints with free, or relatively free motion.

In diarthroses the embryonic tissue uniting the skeletal parts does not remain uninterrupted. It splits, giving rise to a space. The formation of the cavity begins at the sides and extends toward the center of the joint, between the ends of future bones and for a variable distance around them (3). Several clefts thus formed converge into a continuous cavity. Prior to this, the condensed mesodermal forerunner of future bones undergoes changes preliminary to ossification. In the center of what later becomes the shaft of the bone, the dense mesodermal tissue turns into cartilage and then into bone. The extremities, the future articular ends, though as yet cartilaginous, expand and assume definite forms. The configuration of the articular ends of bones and the manner in which the apposing joint surfaces are reciprocally shaped and received determines the variety of diarthroses produced as well as the extent of motion permitted.

THE GENERAL ANATOMY AND THE PATHOLOGICAL PHYSIOLOGY OF DIARTHROSES

Save for the temporary synchondroses uniting the epiphyses and diaphyses of long bones and the articulation between the bodies of vertebræ, the pathological liability of synarthroses is practically nil. On the other hand, diarthroses are prone to a wide range of disturbances. As a matter of fact, when we speak of joints, we tacitly refer to diarthroses and hardly think of synarthroses. Because of their mobility, their comparatively complex mechanics, and the fact that they are composed of several highly differentiated struc-

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tures, such as the articular cartilage or the synovial membrane, diarthroses are subject to a great variety of derangements.

THE COMPONENTS OF THE JOINTS

A diarthrosis consists of a cavity surrounded by synovial membrane and articular cartilage which are supported by the fibrous capsule and the articular ends of bones, respectively. Except perhaps early in disease none of the constituents of the joint is involved to the exclusion of others. But the behavior of various components is singular and the complex phenomenon of joint disease can perhaps be understood better by a consideration of each and every element singly.

The articular cavity. The space contained in a joint can hardly be regarded as one of its components. Yet the articular cavity is a characteristic feature of diarthroses. Upon its integrity depends the function of the joint, as movements are compromised when the joint space is invaded by adhesions and exudates, or when it is encroached upon by overgrowths from the surrounding tissues.

The articular cavity is bounded by the synovial component of the joint capsule and by the cartilage covering the ends of bones. The cavity is variable in extent; in some joints, such as the sacro-iliac, it is almost nonexistent; in others, the knee for instance, it is large. The capacity of a given joint cavity varies with disease and, to some extent, with age. The cavity may be completely obliterated, or it may be broken up into smaller spaces by adhesions. Due to the extensibility of the joint capsule, after repeated effusions the cavity may attain great dimensions. There is normally a partial intra-articular vacuum and joints are under negative pressure. When the joint is filled with fluid, the tension within the cavity becomes positive and may reach as high a level as 700 millimeters of water (32).

Bursæ in the vicinity of the joint may connect with the articular space, and adventitious communications may be established incidental to intra-articular fractures, dislocations with capsular tear, in osteomyelitis complicating the joint, and when pus breaks into or out from the joint cavity. Normally, however, the joint cavity, as visualized by a radio-opaque substance, casts a confined shadow that has a smooth, unbroken contour. When there are adhesions between the apposing surfaces, the contour of the cavity, similarly visualized, will be jagged and irregular. With the same technique, pedunculated fat pads and hypertrophied villi bulging into the joint space, sinuses leading into and out from the joint, and other variations in the content, capacity, and the contour of the articular cavity may be demonstrated (Figs. 1 to 7).

The articular cartilage. On the side toward the articular ends of bones, the joint cavity is bounded by hyaline cartilage. The cartilage covers the ends of the bones; toward the cavity it presents a smooth, glistening surface. The cartilage is essential for the maintenance of joint space and the gliding, insensible execution of movements. Being somewhat elastic, it serves as a resilient cushion, absorbs shock, disseminates stress and strain, and protects the underlying bone from pressure. Structurally (13) the cartilage consists of hyaline matrix and cells which are enclosed in lacunæ. The cells nearer the joint cavity are flattened in a plane parallel with the surface; those in the intermediary zone are disposed irregularly, and the deep cells are arranged vertically. The interstitial substance is considered a product of the cells and the capsule around the cells is interpreted as the matrix most recently laid down. Notwithstanding its homogeneous appearance, hyaline matrix contains collagenous fibrils which are arranged with adaptation to the mechanical needs of the cartilage. On the side where the cartilage is bounded by bone, the matrix is infiltrated with calcium salts, and the line of demarcation between cartilage and bone is relatively sharp (Fig. 8).

The articular cartilage has no nerves, lymphatics, or blood vessels of its own. Pain is incited indirectly by pressure upon the sensory endings in the underlying bone, or within the surrounding capsule. Nourishing fluids reach the interior of the cartilage by transference through the bloodless matrix. Three sources of nutrient supply have been suggested: the synovial fluid (25), the blood vessels of subchondral bone (35), and the

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 7.

Figs. 1 to 7. The articular cavity. Arthographs showing variations in the contour, the content, the capacity, and the communications of the joint space.

Figs. 1 and 2. Normal visualization of the knee joint.

Fig. 3. Adhesions.

Fig. 4. Outpouchings or diverticula.

Fig. 5. Intrusions by synovial overgrowth.

Fig. 6. Increase in capacity due to repeated effusions and also extensive villous encroachment in suprapatellar region.

Fig. 7. Sinuses leading out from the hip joint.

rich network of "circulus articuli vasculosus" (15) of the joint capsule. It is said that the superficial stratum of the central articular cartilage thrives upon the synovial fluid; its deeper zone depends upon the lymph exuded by the vessels of subchondral bone, and the marginal cartilage derives its nourishment from the "circulus articuli vasculosus" (6).

Elsewhere in the body, in the ventral ends of ribs for instance, hyaline cartilage is invested with a connective tissue coating, known as perichondrium, which contains chondrogenic cells. After birth, the articular cartilage is bare over its greater central surface and only a narrow slip of the synovial membrane strays over its rim. This marginal covering is said to possess chondrogenic powers (6). The proximity of the rich network of capsular vessels further enhances the regenerative chances of the marginal cartilage, while the central portion has little or no power of regeneration. This does not mean that its wounds do not ever heal. Defects of the central cartilage may be effaced by connective tissue growing from the synovial membrane and from the subchondral marrow spaces; mature cartilage cells may revert toward their precursors, the fibroblasts, and lay down new connective tissue. The latter process is extremely slow and cannot be depended upon. Superficial abrasions of the central articular cartilage may take a long time to heal, or remain unhealed indefinitely, while a deeper gash, which communicates with the cancellous spaces, is soon spanned by the vascular connective tissue of the marrow. Marrow connective tissue is inactive in instances where the articular end of the bone has already undergone necrosis due to vascular interruption, such as occurs in some cases of complete intracapsular fracture of the femoral neck (33).

On the whole, the articular cartilage is relatively inert and plays a passive rôle in disease (31). It is affected by circulatory changes which curtail its nutrient supply; it is eroded and undermined by tufts of connective

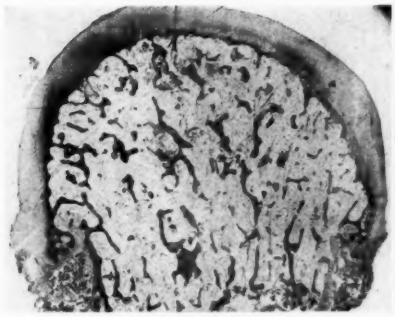


Fig. 8.

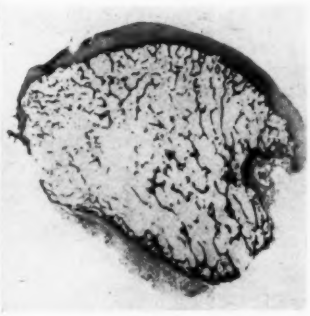


Fig. 9.

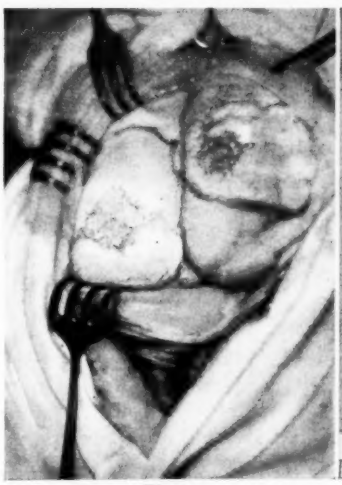


Fig. 10.

Fig. 11.

Figs. 8 to 11. The articular cartilage.

Fig. 8. Photomicrograph of the distal articular end of the metatarsal bone showing the normal relation of the articular cartilage to the subchondral bone and the marginal wedge of synovial membrane.

Fig. 9. Section through a marginal "spur."

Fig. 10. Photograph of the knee at the time of the arthrotomy showing the ulceration of the central articular cartilage of the medial femoral condyle and of the everted patella.

Fig. 11. Resected metatarsal heads from a case of bilateral hallux valgus demonstrating the erosion of the articular cartilage when forced to function in a mechanically disadvantageous position.

tissue growing from inflamed synovial membrane; it is lifted and loosened by subchondral granulations sprouting from the marrow spaces; it cracks and collapses when its bony support breaks down. Trauma, and trauma alone, may be regarded as the primary affection of the articular cartilage. Trauma may be acute or chronic. Contusions of the articular cartilage may heal without symptoms; or the abraded region may manifest surface fibrillation and death of the cells around the contused area (16). A piece of cartilage may break loose into the joint. When both cartilage and bone are free in the joint cavity, the cartilage cells retain their vitality whereas the majority of bone cells die (6). By virtue of the nourish-

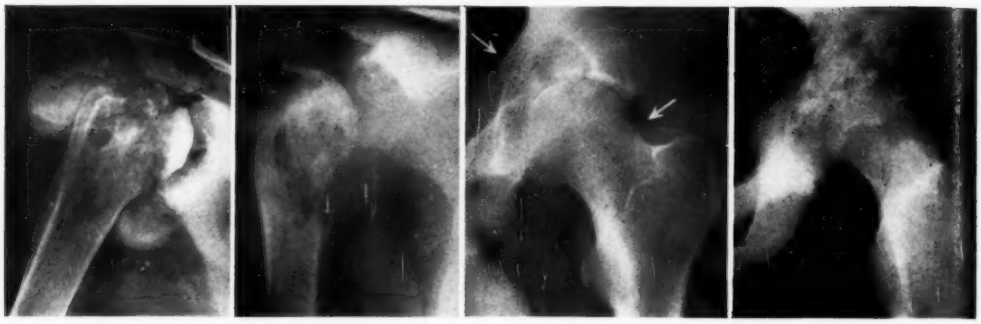


Fig. 12. Fig. 13. Fig. 14. Fig. 15.

Figs. 12 to 15. The articular ends of bones, necrosis due to infection.

Fig. 12. Tuberculous destruction of the shoulder. Note the dense sequestrum, the invasion of metaphysis of the humerus, and the radio-opaque exudate.

Fig. 13. Tertiary syphilitic destruction of the shoulder.

Fig. 14. "Kissing sequestra" of the hip. Note the oval focus in the neck of the femur.

Fig. 15. Diffuse pyogenic destruction of both acetabular and femoral articular surfaces. Note that the destruction present is most extensive at the points of contact and pressure.

ment they obtain from the synovial fluid, the cartilage cells may proliferate and cover their core of dead bone completely; or the broken piece may gain attachment to the synovial membrane and derive additional nourishment through a vascularized pedicle.

Instances of chronic trauma to the articular cartilage are seen when a normal joint is subjected to repeated injuries from incongruity of articular surfaces, from overuse, and from abnormal function. The cartilage wears down at points of contact and pressure; it loses its surface gloss, its resilient hardness, becomes soft, felt-like, velvety. The layer of cartilage nearest the joint cavity slowly disappears and the main body of deeper cartilage splits perpendicularly to the surface. The zone of provisional calcification cracks; the unprotected subchondral bone is exposed, and it wears smooth by usage. The cartilage in the vicinity of the denuded area, especially in the marginal portion, undergoes compensatory hypertrophy and proliferation. Proliferating cells are usually arranged in clumps and lead to thickening of the involved areas. When these areas enlarge and visibly bulge out, they are called ecchondroses. The distinction is made between the commoner marginal ecchondroses and the less frequent nodules beneath the articular surface, which are called epiarticular ecchondroses. Epiarticular swellings tend to produce incongruities of the joint surfaces; marginal growths widen the surface. Ecchondroses may become colonized by the blood vessels and receive an osseous core; then they are called chondro-osteophytes. When ossification is complete they are given the name osteophytes. Ecchondroses, chondro-osteophytes, or osteophytes, may become detached, and when they are free in the joint cavity they are called osteocartilaginous loose bodies (Figs. 9 and 10).

The articular cartilage is also affected by physiological discrepancies. In amputations through the joint, the cartilage at the end of the stump subsequently undergoes connective tissue dedifferentiation; in long standing, unreduced dislocations, the cartilage often passes through a similar regressive change; in an experimentally planned operation when the joint cartilage is forced to function against a tendon (37), rather than cartilage, it wears down and degenerates (Fig. 11). Thus the articular cartilage needs healthy cartilage on the apposed surface to live, and though lack of motion does not in itself cause an undamaged cartilage to die, in prolonged immobilizations the synovial fluid is scant, and being thus deprived of one of its sources of nourishment, the cartilage may degenerate.

The chain of events initiated by erosion and necrosis of a portion of the cartilage with compensatory overgrowths of other areas has been variously called degenerative, hypertrophic.

Fig. 16.

Fig. 17.

Fig. 18.

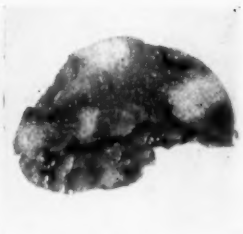


Fig. 19.

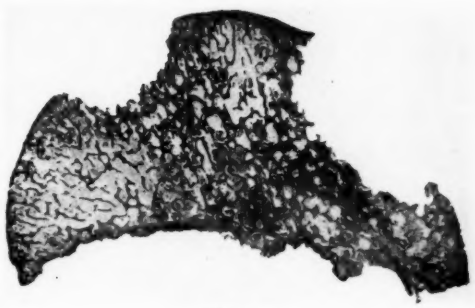


Fig. 20.

Figs. 16 to 20. The articular ends of bones, epiphysiolysis due to infection.

Fig. 16. The slipped epiphysis as the result of pyogenic destruction of the femoral neck.

Fig. 17. The same as Figure 16, 6 weeks later. With traction the shaft of the femur has been pulled down. Note the relative density of the dead femoral head and the shelf of osteophyte growing from the upper acetabular margin.

Fig. 18. The sequestrated epiphysis which was removed surgically; photograph showing the greater destruction of the articular surface at points of contact and pressure.

Fig. 19. Photograph of sagittally sliced surface.

Fig. 20. Same as Figure 19. Photomicrograph of section showing the excavation into the interior of the epiphysis and the extensive necrosis of trabeculæ.

or osteoarthritis. It has experimentally been shown that if a portion of the articular cartilage is injured, removed, or the mechanics of the joint are so altered that function will damage the articular cartilage by incurring repeated trauma, pathological changes characteristic of degenerative arthritis develop when the animal continues to use the injured joint, and such changes do not develop when the joint is immobilized (17).

The articular ends of bones. The supportive framework of joints consists of reciprocally shaped and received ends of two or more bones. The articular ends are composed of cancellous interior and a thin coating of compact bone. The latter is known as the articular lamella, or cortex, and forms the joint surface of the bone. It has no haversian canals, no canaliculi, nor is it penetrated by blood vessels (12). Therefore, it is denser and harder than ordinary compact bone and serves as an unyielding support to the articular cartilage. The cancellous interior of the articular ends contains numerous intercommunicating spaces which are filled with marrow and are bounded by anastomosing trabeculæ. The arrangement of the trabeculæ is such that maximum strength is secured with minimum material: where stress is great the trabeculæ are thicker and more closely welded; in regions of light stress, the trabeculæ are thin and widely spaced. The trabeculæ are invested by a thin layer of connective tissue, the endosteum, which contains bone-forming and bone-destroying cells. The endosteum is so intimately related to the stroma of the marrow that the 2 are regarded as identical by some (5) and are spoken of as the connective tissue elements of the cancellous spaces or of the marrow. There are nerves, lymphatics, arteries, and veins in the marrow and, in addition, marrow contains numerous thin walled capillaries known as sinusoids (23). These are not lined with endothelium but with the flattened cells of marrow stroma; the circulating blood is thus brought into close relation with the supportive and cellular elements of the marrow. The cells of the marrow can pour into the blood stream with phenomenal rapidity, and foreign particles floating in the blood easily penetrate the thin walls of the sinusoids and find their way into the marrow tissue.

The articular ends of bones are thus composed of osseous elements on the one hand and

Fig. 21.

Fig. 22.

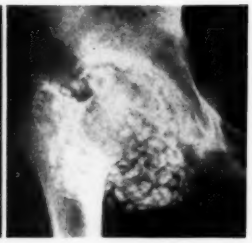


Fig. 23.

Fig. 24.

Fig. 25.

Figs. 21 to 25. The articular ends of bones, aseptic necrosis.

Fig. 21. Dead head incidental to intracapsular fracture of femoral neck.

Fig. 22. Sequestrated head following aseptic epiphysiolysis. Note that the mesial and inferior portion of the dead head is undergoing "creeping replacement," probably by the connective tissue elements of the round ligament.

Fig. 23. Mushrooming and crumbling of the femoral head into numerous osteocartilaginous bodies. This patient "limped" at about the age of 10. The question of an old Perthes' disease was considered.

Fig. 24. Necrosis of carpal lunate (Kienboeck's disease). Note the area of rarefaction within the articular end of the radius opposite the dead bone. This is not unlike the osteoparotic areas one sees in "degenerative" arthritis.

Fig. 25. Necrosis and collapse of the left tarsal navicular (Koehler's disease). Compare it with the navicular on the right side.

vascular connective tissue on the other. Inflammatory reactions start in the marrow, within the cancellous spaces, and involve the bone secondarily. When its blood supply is cut off, as by the pressure of pent-up pus, edema or intravascular occlusion, the bone dies. Proteolytic ferments in the exudate, and perhaps the activated osteoclasts of the endosteum, enhance bone necrosis; vascular granulations originating from the marrow absorb the dead bone at its periphery and separate it from the living (28). The calcium content of the dead bone remains unchanged; but if the necrotic fragment is small, it can be completely digested leaving instead a cavity. The broken down trabeculæ further liberate marrow connective tissue; granulations work their way toward the joint or into the periphery. When the articular cortex is eroded the overlying cartilage detaches and dies. The joint is now invaded by exudate and granulations which attack the opposite articular surface. Denuded bones contact; granulations from both sides meet, unite; adhesions are formed. When inorganic salts impregnate the connecting granulations, bony ankylosis is the outcome. Proliferated connective tissues which work their way peripherally toward periosteum lay the foundation for marginal "spurs." The virulence of the organism, the

Fig. 26.

Fig. 27.

Fig. 28.

Fig. 29.

Fig. 30.

Fig. 31.

Figs. 26 to 30. The articular ends of bones, incongruity arising from miscellaneous causes.

Fig. 26. Pyogenic invasion of upper tibial epiphysis with collapse of the medial articular cortex.

Fig. 27. Partial arrest of epiphyseal growth resulting in tilting of the tibial plateau and knock-knee.

Fig. 28. Unevenness of tibial surface due to malunited, depressed fracture of lateral tibial condyle.

Fig. 29. Old united intracapsular fracture of femoral neck. Note the collapse of the articular cortex due to weight bearing before the dead femoral head had been completely replaced and organized.

Fig. 30. Incongruity resulting from lack of appositional remodeling of the acetabulum and femoral head in congenital dislocation of the hip.

Fig. 31. The articular ends of bones. Tumor of the distal tibial articular end at the ankle joint. Biopsy was reported as "spindle-cell" sarcoma and it was thought to be a malignant change coming on in a previous giant cell tumor. Note the comparatively intact articular cortex.

character of the exudate it incites, and mechanical factors of contact and pressure within the invaded joint variously modify the picture (Figs. 12 to 15).

Articular ends may break down due to aseptic necrosis. This may result from injuries, vascular blockage, overexposure to radium or x-ray, and by the nitrogen gas of caisson disease. There are a number of related conditions—as Perthes' disease of the hip, Koehler's of the tarsal navicular, and many others—in which areas of necrosis of the

Fig. 32.

Fig. 33.

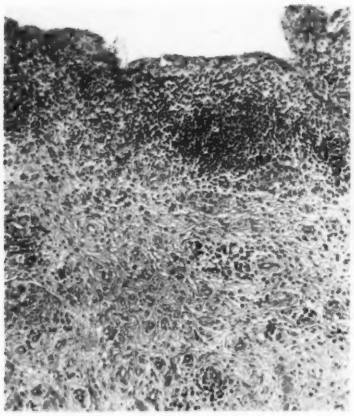


Fig. 34.

Fig. 35.

Figs. 32 to 35. The synovial membrane.

Fig. 32. Photograph of surgically opened knee joint of a young girl suffering from "proliferative arthritis." Note the synovial pannus invading the articular surfaces of both femoral condyles—especially the external—and the pearly egg-shaped loose body beneath the medial condyle.

Fig. 33. Same as Figure 32, showing the erosion of the articular cartilage underneath the synovial pannus which was wiped away.

Fig. 34. Section through thickened membrane demonstrating agglomeration of round cells underneath the synovial surface and around the blood vessels.

Fig. 35. Photomicrograph of a section from another case showing the enlarged villi.

growing bone lead to eventual malformation of the articular ends. In certain joints, such as the hip, the plate of cartilage connecting the epiphysis and diaphysis lies within the capsule. Disturbances of this cartilage may lead to slipping of the articular end with consequent misalinement with the shaft. In pyogenic coxitis, the epiphysis is sometimes completely cut off from its main blood supply and dies (Figs. 16 to 20). The same holds true in a certain percentage of intracapsular fractures of the femoral neck (29). Vascularized connective tissue may invade the dead head by way of the round ligament, through

Fig. 36. Fig. 37. Fig. 38. Fig. 39.

Figs. 36 to 40. The synovial membrane.

Fig. 36. The opaque oval mass in the lateral aspect of the elbow was thought to be calcification within the radiohumeral bursæ. On biopsy it was found to be a nodular thickening within the synovial wall which consisted of strands of collagenous fibers and connective tissue cells.

Fig. 37. Osteocartilaginous bodies which were presumed to originate from the synovial membrane since the skeletal articular surfaces of the knee are smooth and unbroken.

Fig. 38. Calcification within the synovial wall, in the infrapatellar fat pad.

Fig. 39. Synovial cyst visualized by injection of iodized oil.

Fig. 40. The surgically removed specimen of the same together with the medial semilunar cartilage.

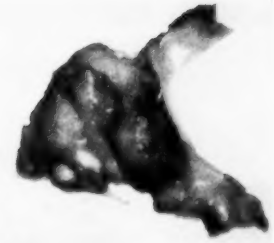


Fig. 40.

untorn portions of the capsule, or through attachments formed between the two articular surfaces. The invading connective tissue replaces the dead marrow and bone and may eventually organize into new bone by metaplasia, which takes an appreciable time to complete itself. When weight is borne upon a dead or incompletely substituted articular end, its cortex collapses, the overlying cartilage caves in, and the joint surface becomes irregular, uneven. Incongruities of the articular surfaces also arise from unreduced or improperly reduced fractures into the joint (Figs. 21 to 29).

Articular ends are capable of functional adaptation. Alterations in the mechanics of the joint are reflected by changes in the internal architecture as well as in the external configuration of the bone. At points of excessive pressure the trabeculæ thicken and increase their lime content; where no weight is borne, or stress due to motion is eliminated, the trabeculæ thin and rarefy. Lack of appositional stimulation retards the development of both femoral head and acetabulum in congenital dislocation of the hip (Fig. 30). The acetabulum remains shallow; the small femoral head may undergo such adaptive changes when forced to function in abnormal situations for a long time that, even when put into a deepened acetabulum, its shape and alinement with the shaft may be incompatible with normal hip function (2). Tumors of the articular ends of bone may eventually break into the joint though the cartilage is very resistant to such invasions (Fig. 31).

The joint capsule. The sleeve of connective tissue which envelopes the joint cavity and binds the skeletal parts together consists of an outer coating known as the fibrous capsule and an inner lining, the synovial membrane. The fibrous capsule is irregularly reinforced by thick bands and is supported by tendons, muscles, and ligaments. In its turn it supports the synovial membrane. In some joints the fibrous capsule is incomplete and the synovial membrane rests on fat and areolar tissue, muscles, and periosteum. In the same joint, there are areas in which the fibrous capsule and the synovial membrane are inseparably

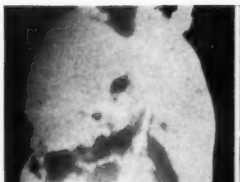
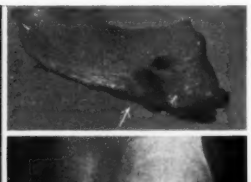


Fig. 41.



Figs. 42 and 43.

Figs. 41 to 43. Fibrocartilages.
Fig. 41. Arthrography of the knee. Note the zigzag line passing through a tear at the chondrosynovial junction of the medial meniscus.
Fig. 42. Vascularization of untorn, chronically injured semilunar cartilage.
Fig. 43. Calcification of medial meniscus of the knee.

fused; at other points, the interposition of fat and areolar tissue makes it possible for the synovial lining to be lifted as a separate layer.

Fibrous component. The fibrous capsule connects the articular ends of bones, restricts motion, and helps to stabilize the joint. Severe trauma may rupture the capsule and, unless the torn edges are carefully apposed and held together, there remains a gap through which the synovial membrane may herniate. A similar situation obtains in the aged and arthritics in whom the synovial membrane bulges through worn-out areas as does an inner tube through the frayed spots of an old casing. After repeated effusions, or incidental to chronic strain due to overweight, in dislocations and paralytic subluxations, the capsule becomes lax. Conversely, it may undergo contractures secondary to inflammatory diseases of the joint, to malformations, to burns and cellulitis, or to upper motor neuron lesions. Lastly the fibrous capsule may form tumors.

The synovial membrane. The synovial membrane lines the articular aspect of the joint capsule. Toward the joint cavity it presents a pale-gray, moist, glistening surface which bears a superficial resemblance to serous linings; but there the resemblance ends. Serous membranes line the primary cavities that originate from the celom early in embryonic life; joints are developed later from mesoderm. Serous membranes are lined by a regularly patterned layer of cells, the endothelium; the cells of the synovial membrane may lie on the surface but do not line it in any orderly fashion. Serous surfaces appear smooth and are smooth; synovial surfaces may appear smooth but closer observation will reveal tags, villi, and pleats. Finally, synovial membranes differ from serous coatings by their relative thickness, their richness of blood vessels and their slimy secretion, which makes them functionally akin to mucous membranes. Structurally, synovial membranes have nothing more in common with mucous linings than they have with serous coatings.

The synovial membrane is a modified connective tissue lining a connective tissue space, the joint cavity. It is attached around the margins of the articular cartilage, from the edges of which it sweeps over the cartilage-bare intra-articular ends of bones; it is then reflected to line the fibrous capsule. Tendons and ligaments within the joint are invested by the synovial membrane, and cartilages receive a slip of marginal coating from it. In adaptation to the mechanical conditions to which the surface of a given area is subjected, the synovial membrane presents a

Fig. 44.

Fig. 45.

Figs. 44 and 45. Ligaments.
Fig. 44. Calcification of previously injured collateral ligament of the knee (Pellegrini-Stieda's disease).
Fig. 45. Freshly torn fibular collateral ligament causing lateral instability of the knee.

variable structure (18). Areolar synovial membrane lines those parts of the joint where movement of the surface over the underlying tissue is needed; the fibrous type covers the areas which are subjected to pressure or strain as over intra-articular ligaments and tendons; the adipose variety invests the fat pads which project into the joint cavity. From within outwards, the membrane consists of two strata (6, 31): an internal and an external. The internal is compact, cellular, and, on the whole, relatively avascular; the external is richly vascular and, save in the fibrous type of synovial membrane, it is loosely constructed and contains a variable amount of fat cells and areolar spaces. Of special interest is the area of transition between the synovial membrane and the articular cartilages. As the cartilage is approached the synovial membrane splits into a wedge, the base of which roughly corresponds to the margin of the cartilage. (Fig. 8); the surface cells flatten and gather into groups simulating cartilage cells in lacunæ; as such a strip of synovial membrane strays over the rim of the cartilage. Within the folds of the synovial wedge, around the circumference of the cartilage, is the rich network of capsular vessels. Somewhat more peripheral, stemming from the synovial membrane proper, are the villi, pleats, and fringes, which are pedicled projections of the membrane: they produce a tremendous increase in the absorptive surface of the joint capsule (22). Besides blood vessels, synovial membranes contain nerve endings and lymphatics. The blood vessels do not lie bare on the surface of the synovial membrane: a few cells or a strand of collagenous fibers separate them from the joint cavity.

Fig. 46.

Fig. 47.

Figs. 46 and 47. Muscles and tendons.
Fig. 46. Posttraumatic myositis ossificans about the elbow incident to dislocation.
Fig. 47. Calcification of supraspinatus tendon.

Fig. 48.

Fig. 49.

Figs. 48 and 49. Bursæ.
Fig. 48. Visualization of the subacromial and subdeltoid bursæ.
Fig. 49. Arthrography demonstrating the continuity of the popliteal bursa with the cavity of the knee.

The function of the synovial membrane is to provide the joint with a smooth, flexible surface; it is in part responsible for the production of synovial fluid. Though well substantiated proofs are as yet wanting, it is generally conceded that the synovial membrane plays an important, perhaps active, rôle, in the exchange of material between the joint cavity and the circulation. Crystalloids are quickly removed from the articular cavity; suspensions of fine colloids diffuse through the synovial membrane into the subsynovial tissue and thence into the blood stream and lymphatics; a coarse colloid is removed from the joint with more difficulty (19). Gross particles like a fragment of bone may become surrounded by the synovial membrane in a fold of adhesion or gain an attachment to its inner aspect and acquire a crust of new cartilage covering its raw surfaces. The membrane can itself produce osteocartilaginous loose bodies. The synovial membrane regenerates when dissected away (20).

Structurally, functionally, and pathologically, the internal stratum of the synovial membrane has been regarded as analogous to the articular cartilage, while the external layer is considered akin to the cancellous spaces of the articular ends of bones (31).

Fig. 50.

Fig. 51.

Fig. 52.

Fig. 53.

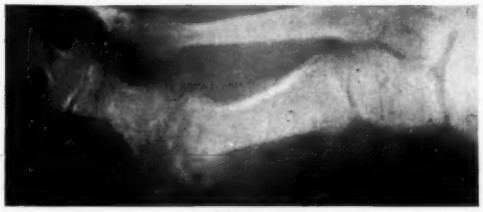


Fig. 54.

Figs. 50 to 54. Neurogenic joint disturbances.

Figs. 50, 51, 52, and 54. Tabetic arthropathies of the hip, knee, ankle, and of the big toe. Note the sequestration of the femoral head shown clearly in Figure 50.

Fig. 53. Dislocation of the hip due to anterior poliomyelitis.

Together with the cartilage the internal stratum provides the joint with a gliding surface and preserves the integrity of the cavity; like the articular cartilage, in disease it plays a comparatively passive rôle. On the other hand, the loosely constructed and richly vascular external stratum becomes the active arena of inflammatory changes: hyperemia, edema, cellular infiltration, and tissue proliferation. From the loose areolar spaces of the external layer, tissue fluids pass into the joint cavity giving rise to hydrarthrosis, which lasts as long as hyperemia persists. If irritation continues, prolonged congestion of the external layer leads to cellular infiltration and tissue proliferation. Finger-like processes of proliferated connective tissue indent the internal stratum and project into the articular cavity creating a phase of joint inflammation known as chronic villous synovitis. Pannus is a connective tissue column which has crept over the articular cartilage, has adhered to its surface. Vascular granulations invading the joint cavity attack and erode the articular cartilage and establish adhesions between the joint surfaces. Usually proliferative activity is going on in the marrow connective tissue at the same time and the articular cartilage may be attacked from below as well as from its margins. When the articular cartilages are completely eaten away, the granulations contact to form adhesions; with subsequent ossification of these connecting strands, bony ankylosis is the outcome. The slow culmination of these events, because of superficial clinical resemblance to rheumatic fever, has been given the name rheumatoid arthritis (8); frequent, though by no means exclusive, association of the joint lesion with foci of nonspecific infections has given others cause to coin the term infectious arthritis, and when such foci are not manifest, the adjective atrophic is substituted, since, on opening the joint, the cartilage is seen frayed and x-ray films show the bones to be porotic (11); however, proliferation of marrow and synovial

Fig. 55.

Fig. 56.

Figs. 55 and 56. Circulatory relations.
Fig. 55. Immediate lymphatic drainage from the knee. Note the popliteal node and the efferent and afferent tracts.
Fig. 56. The swollen, stiff knees of a patient suffering from thrombo-angiitis obliterans.

connective tissue is regarded as the basic pathological feature, and proliferative arthritis is a term preferred by some (24) (Figs. 32 to 35).

The internal stratum of the synovial membrane, though permeable to tissue fluids, at first resists the infiltration of cellular elements within the external layer (31). In time, however, with persisting edema and congestion of the external stratum, this barrier is broken, and exudate pours into the joint cavity. If bacteria are lacking, aseptic pyarthrosis is the result; with infection, septic pyarthrosis ensues.

Tears of the synovial membrane which involve both the internal and external strata are usually followed by hemarthrosis. Fresh blood in the joint, undiluted with synovial fluid, clots easily; but after it has been attacked by inflammatory exudate it remains liquid, though it undergoes disintegration (31). The red blood cells break up and donate their pigment to the joint fluid while the fibrin may organize into adhesions. Blood clots in the external stratum, especially in fat tissue, may calcify and even ossify.

The synovial membrane is capable of producing new-growths. Reference has already been made to the production of synovial chondromas. Synovial cysts are areas of mucoid degeneration within the membrane. The synovial membrane may form xanthomatous growths. "Synovioma" is a term limited to tumors arising from the "lining mesothelium" (4), which probably refers to the internal stratum of the synovial membrane (Figs. 36 to 40).

Synovial fluid. The origin of the small amount of watery, viscid liquid normally contained in the joint cavity is not as yet definitely established. Accepted opinion leans toward regarding this fluid as partly a secretion by the "special" cells of the synovial membrane and in part a transudate from the circulation (22). The fluid is alkaline in reaction and contains small amounts of mucin, albumin and extractives, fat and inorganic salts (18). Pending further investigation the following characteristics, gleaned from the literature (6, 7, 21, 22) may be regarded as normal or nearly normal: specific gravity, 1.040; viscosity, 10.7–20.0; hydrogen-ion concentration 8.2–8.4; total solids, 4.4 per cent; total protein, 1.6 per cent; mucin, 1.95 per cent; bilirubin, not exceeding 0.5 milligrams per cent; sugar, 80–120 milligrams per 100

Fig. 57.

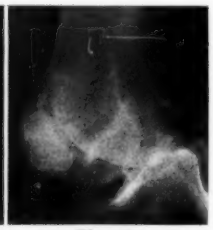


Fig. 58.

Figs. 57 to 61. Mechanical disturbances.

Figs. 57 and 58. X-ray films of the knees of a man whose weight in life varied between 300 and 380 pounds.

Fig. 59. Photograph of the femoral articular surface obtained at necropsy. Note the ulceration of the central articular cartilage, and marginal thickenings. Erosion of the joint surface is more extensive over the medial condyle since it bears the brunt of heavy weight. Note also the osteocartilaginous body ready to detach from the intercondyloid fossa.

Fig. 60. The corresponding tibial surface.

Fig. 61. Photograph of the lateral view of the distal articular end of the femur showing marginal lipping.

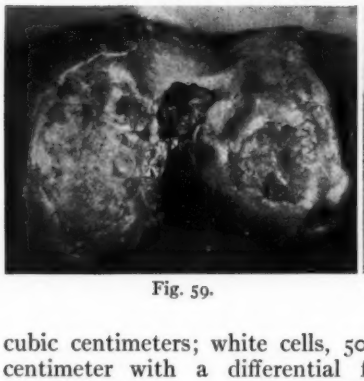


Fig. 59.

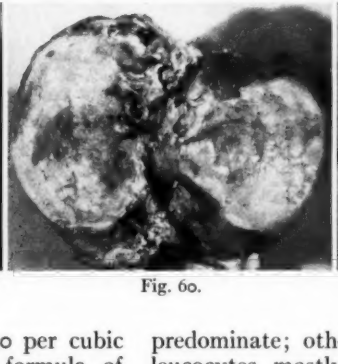


Fig. 60.

Fig. 61.

cubic centimeters; white cells, 50 per cubic centimeter with a differential formula of mesothelial cells 3 per cent, granulocytes 5 per cent, macrophages 30 per cent, and monocytes 58 per cent; there may be as many red cells as white. The function of the synovial fluid is threefold: it lubricates the joint surfaces and thereby facilitates motion; because of its alkalinity it protects the articular cartilage against acid metabolites; it helps to nourish the articular cartilage. Motion is a physiologic stimulus for the production of a normal amount of synovial fluid.

In disease the joint fluid changes in quantity, composition, chemical and immunological reactions. The largest effusions are seen in severe inflammations and in acute disrupting trauma with hemorrhage into the joint cavity. In chronic joint diseases, the effusion is usually small. When the aspirated fluid is acid and its glucose content is down to 60 per 100 cubic centimeters or lower, one suspects septic arthritis. Shift of hydrogen-ion concentration toward alkalinity on repeated aspirations denotes favorable prognosis. In acute exudates from trauma or infection, the total cell count of the fluid is increased; in hemarthrosis the red blood cells predominate; otherwise the cells consist of leucocytes mostly. A leucocytic count of 11,000 or more per cubic millimeter of synovial fluid which is associated with 60 per cent or more of granulocytes, in a case of chronic non-specific joint disease, is likely to be accompanied by positive culture of attenuated organisms (7). The culture in septic arthritis yields ready growth of one of the following bacteria: streptococci, staphylococci, pneumococci, and rarely meningococci, Bacillus typhosus, Brucella melitensis. In gonorrheal arthritis, the culture may be positive and the complement fixation test of the synovial fluid may corroborate the blood findings (22). Guinea pig inoculation of the joint exudate in a case of suspected tuberculous arthritis may confirm the diagnosis. Smears of the synovial exudate are usually positive in septic arthritis, at times in advanced tuberculosis and occasionally in acute gonorrhea. In syphilitics the synovial fluid gives a positive Wassermann reaction when the blood gives a positive reaction, but this does not necessarily mean that the joint lesion is luetic; however, a positive reaction yielded by the joint fluid in the presence of a negative blood Wassermann is regarded as an evidence of syphilitic arthri-

tis (7). In jaundice the icterus index of the synovial fluid is increased, but an icterus index of over 5.5 without generalized jaundice signifies a recent traumatic arthritis or subsiding hemarthrosis. The presence of fat globules in the aspirated fluid makes one suspect a tear involving the subsynovial adipose pads or intra-articular fractures putting the joint in communication with the bone marrow. An increase in cholesterol content of the joint fluid would be suggestive of xanthomatous tumor of the synovial membrane (4).

Pathological joint exudates contain ferments. They may come from bacteria, from dead tissues, or from the leucocytes. The tubercle bacillus is said to contain no proteolytic ferments, its toxins destroy the autolytic enzymes of dead tissues; the leucocytes contained in tuberculous granulations or exudate are mainly mononuclears; their ferment is active in acid media, but not in tuberculous pus, which is alkaline in reaction (26). Pyogenic exudate is acid and it contains an abundance of polymorphonuclear leucocytes which produce proteolytic ferments. Whereas the digestive action of tuberculous joint exudate is practically nil, that of pyogenic pus is marked. Proteolytic ferments assist greatly in rapid dissolution of necrotic cartilage in pyogenic arthritis; cartilage in tuberculous arthritis may persist months or years (30).

Accessory structures. Fibrocartilages enter into the formation of some joints. In the hip and shoulder they are found as circumferential bands, the labrum glenoidale, which deepens the articular sockets; in the temperomandibular, sternoclavicular, acromioclavicular, and wrist joint, a biconcave plate, the articular disc, subdivides the cavity; in the knee, two crescents, the similunar cartilages, protrude into the joint from the lateral and medial sides. In general the fibrocartilages obliterate the space between apposed articular surfaces, promote better reception of one articular end into the other, absorb and disperse their share of shock and pressure and in a measure modify the movements of the joint. It is possible that the interarticular fibrocartilages act as slippery fulcrums and play a rôle in the frequent dislocation of the temperomandibular, acromioclavicular and shoulder joints; perhaps it is not coincidental that gonorrheal arthritis often settles in the knees, wrists, and sternoclavicular articulations, though joints without fibrocartilages are not immune to this infection. On the whole, however, fibrocartilages in freely movable joints other than the knees are not definitely known to cause or create any disturbance. The two menisci of the knee may loosen from their moorings, become displaced, tear or split following trauma and may thus usher in a chain of events known as internal derangements. Occasionally the semilunar cartilages may undergo calcification, even ossification; at times they form cysts. Interarticular fibrocartilage lesions mechanically interfere with function of the joint and give rise to frictional erosions of articular surfaces (Figs. 41 to 43).

Fig. 62. Fig. 63.

Figs. 62 and 63. Primary germ cell affinity. Drawings of the hands of a man with "proliferative arthritis," subcutaneous nodules, and myocardial heart disease.

Ligaments are flattened bands of white fibrous tissue which bind together the articular ends of bones and help to stabilize the joint. Distinction is made between the capsular ligament, which is another name for the fibrous component of the joint capsule, and the accessory ligaments, which aid the fibrous capsule but stand apart from it (12). Accessory ligaments may be extracapsular, such as the fibular collateral ligament of the knee, or intracapsular, as are the cruciates.

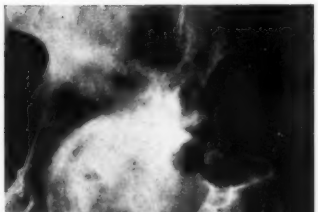


Fig. 64.

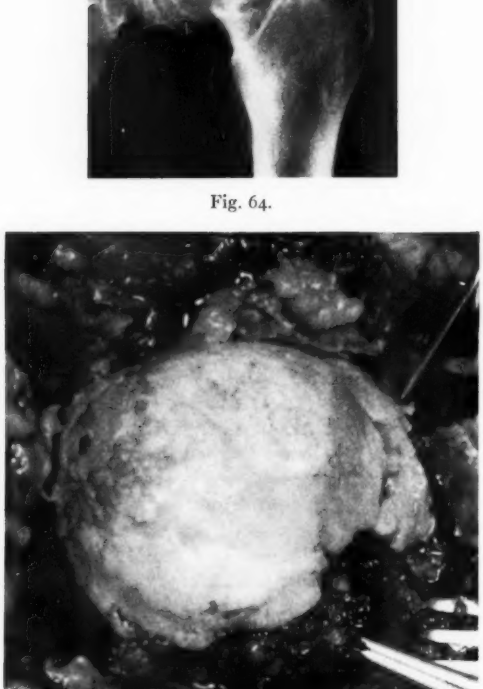
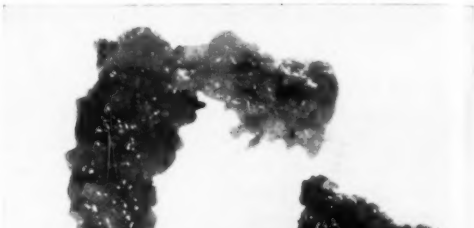


Fig. 65.



Figs. 66 and 67.

Figs. 64 to 67. Inflammatory and mechanical factors combined.

Fig. 64. X-ray picture of the hip of a man suffering from senile hip disease.

Fig. 65. Photograph of surgically dislocated hip. Note the fibrillation of the cartilage at points of pressure and hypertrophy of the margins. Hemostat points to proliferated villous synovial overgrowth. The subcapital cavity seen in the film was filled with fatty marrow.

Fig. 66. Photograph of dissected thickened "villous" synovial membrane from around the femoral neck.

Fig. 67. Photomicrograph of a section through the thickened marginal cartilage superimposed by a layer of new bone and then by newly deposited fibrocartilage.

Intracapsular ligaments are also found in the hips, wrists, talocalcaneal and tarsal joints. The flexibility of the ligaments permits motion at the joint; their great tensile strength resists disruption. When their elasticity is overcome and they are stretched, ligaments are said to be *strained*. In *sprain*, individual fibers tear and the ligament as a whole becomes lax. However, both strain and sprain are clinical terms lacking pathologic verification and probably represent more than one lesion and involve other structures around the joint as well (9). Ligaments are moderately vascular and richly supplied with sensory nerves (31). When injured they are very painful and may be accompanied by regional bleeding—

hemarthrosis in case of intracapsular ligaments. Ligaments may tear through their substance or at their attachment, with or without avulsion of a piece of bone. Months after an injury x-ray films may reveal a calcified shadow coinciding with the position of the ligament. When the ligament is completely torn, the joint loses its stability in part and can be carried through an excessive range of motion. The ruptured ends retract and, unless carefully approximated and sutured, the mechanics of the joint may be disturbed and the articular cartilage may wear away in areas of abnormal stress and pressure (Figs. 44 and 45).

Muscles and tendons. Muscles maintain posture and execute movements. Postural contractions help to hold the articular surfaces apposed; phasic modes produce motion at the joint. The joint itself is passive: it merely permits motion. In both primary and neurogenic disturbances of muscles the joint concerned is secondarily affected: it contracts, stiffens, loses its stability, becomes flail, undergoes subluxation and even breaks down architecturally. In turn, diseases of the joint may incite reflex muscular spasm, causing the muscle and the tendons to undergo atrophy, fibrosis, contracture, calcification, and at times ossification. Certain muscles are more intimately related to neighboring joints than others; then again some muscles have to work in a mechanically disadvantageous situation. The brachialis muscle covers the front of the elbow joint and lies in close relation to the articular capsule; following dislocation and severe injuries to the elbow, myositis ossificans often settles in a hematoma within the muscle or at the point of avulsion. The tendon of the long head of the biceps brachii, enclosed in a special sheath of the synovial membrane, passes through the shoulder joint: lesions of this tendon or its sheath are bound to affect the joint. The muscular fibers of the supraspinatus converge to a tendon which crosses the upper part of the shoulder joint and is intimately adherent to the articular capsule; the muscle contracts in a bony chamber, the supraspinatus fossa; its tendon is constantly compressed against bone immediately proximal to its insertion into the great tuberosity of the humerus; continued trauma induces the muscle to degenerate and its tendon to undergo fibrosis, with calcareous deposits within its substance; abduction forces the calcified mass against the acromion and to avoid pain the arm is kept to the side; contracture of other muscles and joint structures may ensue (Figs. 46 and 47).

Many *bursæ* lie in the neighborhood of joints and some communicate with the articular cavity. They facilitate motion and minimize frictional irritation. Their lining is like that of joints and the reactions of bursal walls resemble those of the synovial membrane. Infections in the bursæ may spread to the neighboring joint (Figs. 48 and 49).

THE JOINT IN RELATION TO THE REST OF THE BODY

There is perhaps no organ in the body which does not directly or indirectly influence the joints. Diseases elsewhere often affect the articulations. One need only mention joint involvement in connection with pulmonary diseases, allergic states, psoriasis, gout, and hemophilia. It has recently been observed that jaundice alleviates arthritic pains (14); vitamins, nutritional factors, and endocrines are gaining increasing attention in literature. Further study may clarify these and numerous other interrelations. At the present all one can say is that joints are provided with a nervous mechanism; they are supplied by arteries, veins, and lymphatics; they are functionally correlated with one another, with bones and muscles; and perhaps, as derivatives of mesoderm, they possess certain endogenous characteristics common to all tissues of that origin.

The nervous mechanism. The articular ends of the bones, the synovial membrane, the fibrous capsule and the accessory ligaments are well supplied with sensory nerves; blood vessels carry sympathetic filaments. Pancinian corpuscles and other nerve endings installed in articular structures register proprioceptive sensations as well as sense of pain and pressure. When these afferent impulses fail to reach the central nervous system there is lack of muscular co-ordination and the joint may be injured unawares. This occurs in

Charcot joints of tabes dorsalis and syringomyelia. Disturbances in the connecting and efferent links of the reflex arc cause joint lesions in their turn, as exemplified by posthemiplegic arthritis or subluxations of anterior poliomyelitis (Figs. 50 to 54).

Circulatory relations. Articular structures contain lymphatics and blood vessels and are nourished by them. Comparative analyses of plasma and synovial fluid indicate very close relationship between the two (27): glucose ingested by mouth will appear in the synovial fluid with surprising rapidity, reaching there a concentration in excess of that of blood; rise of sugar and bilirubin in the blood are followed by corresponding elevations in the synovial fluid; some drugs, like sulfanilamide are recovered in the joint fluid. It is a foregone conclusion that infections and noxious metabolites circulating in the blood may affect the joint; local processes in the joint may give rise to systemic reactions. Circulatory changes have their effect on joint structures: hyperemia is said to cause the bones to decalcify; chronic passive congestion results in hypertrophy of bone (the clubbed fingers of cardiacs); diminished circulation is followed by increased calcification; when the blood supply is completely cut off, the bone dies and its lime content remains static (36). The association of arteriosclerosis and osteoarthritis has long been noted and it has recently been found that a large percentage of patients suffering from rheumatoid arthritis maintain a comparatively low peripheral temperature level. It is suggested that this is due to diminished capillary flow caused by vasoconstriction. Again in this disease there is a delay in the rapidity with which glucose ingested by mouth and absorbed into the blood stream leaves the vascular channels; and this delay, too, is ascribed to systemic vasoconstriction which is said to parallel or precede the onset of arthritis (27). Experimentally, ligation of the blood vessels of the patella is followed by degeneration and hypertrophy of the articular cartilage (38); the patella itself hypertrophies (10) (Figs. 55 and 56).

Mechanical interdependence. Joints are functionally related to one another, to the bones and to the muscles, and are influenced by disturbances of posture and body mechanics in general. Composite movements like squatting and walking require synchronous action at several joints, the hips, knees, and ankles; and a mechanical handicap in one necessitates adaptive changes in the others. Shift of the lines of stress and strain from the usual bearing surface of the joint to areas unaccustomed to great pressure may wear the cartilage at these points and cause the underlying bone to undergo sclerosis and the capsule or the ligaments nearby to become lax. This is often seen in the knees and ankles of the extremely obese (Figs. 57 to 61). Similar incongruities may occur when a neighboring joint becomes stiff or deformed, when there is a malunited fracture in the bones above or below the joint, in partial arrest of epiphyseal growth on either side of the joint, and in consequence of such static deformities as curvatures of the spine, tilted pelvis, coxa vera, knock-knees, bow-legs, and everted or inverted feet.

Primary germ cell affinity. The concurrence of myocardial diseases, subcutaneous nodules, and joint affections in rheumatoid states has been ascribed to simultaneous involvement by certain strains of bacteria from a hidden focus of infection. The question still remains unsettled; but one must not overlook the possibility that, with other derivatives of mesoderm, joints inherit certain constitutional and endogenous peculiarities which make them susceptible to "hyperergic" inflammation (34). The intense inflammatory changes which follow the intra-articular injection of antigen in sensitized animals, involve the joint injected, the arteries and veins about the joint, and the skeletal musculature near and far from the point of injection. Repeated intra-articular injections of horse serum result in deforming and ankylosing joint disease. The local "hyperergic" responses are associated with similar changes in other joints, in the myocardium and in large arteries (Figs. 62 and 63).

SUMMARY AND CORRELATION

A diarthrosis is a movable joint. It consists of the cartilage-covered ends of bones and a capsule which is lined by the synovial mem-

brane. It encloses a cavity and the cavity in turn contains a small amount of lubricating fluid. Accessory structures further complicate the anatomy of some joints and neuromuscular, vascular, and mechanical integrations create added complexities. The joint itself is formed of connective tissue derivatives and it manifests the elemental properties of its mother tissue with but slight modification. In health the function of connective tissue is mechanical; in disease, it is reactive. It supports and connects parts, restrains or permits motion between them. When irritated, it undergoes a local adaptive change characterized by proliferation of cells immediately around the injured area and attraction of wandering cells to the region. Proliferation of cells leads to granulation which, aided by the ferments of the exudate, erodes and replaces the more differentiated elements, and repair takes place by formation of scar tissue. Secondary metaplasia and differentiation may then follow.

Generally speaking, the more undifferentiated a tissue, the more it is apt to undergo proliferation and, conversely, the more highly differentiated a tissue the less is its capacity to proliferate, repair or reproduce itself (1). In joints relatively undifferentiated connective tissue is found in the external strata of the synovial membrane and in the marrow spaces of articular ends. Differentiated elements are represented by the internal layer of the synovial membrane, the articular cartilage, the trabecula, and the cortex of the articular ends of bones, and, to a lesser extent, by the fibrous capsule and accessory ligaments. Moreover, the relative vascularity of the external stratum of the synovial membrane and the marrow spaces favors localization here of circulating irritants; these two regions become the active arena of inflammatory changes. The more differentiated derivatives of connective tissue—bone and cartilage in particular—passively resist inflammation; they bear the brunt of shearing and pressure stresses and break down when their elasticity is overcome. Both cartilage and bone are affected by disturbances of their nutrition which hasten their disintegration. Circulatory and nutritional changes may accompany, or result from, inflammatory processes; exudates and vascular granulations may damage the joint so as to create incongruities between apposed articular surfaces and the joint thereafter becomes liable to self-inflicted injury; in its turn, trauma affecting the skeletal elements primarily may initiate a point of irritation in the undifferentiated derivatives of connective tissue and set them toward inflammatory reactivity (Figs. 64 to 67). Moreover such mechanical factors as points of contact and pressure between apposed articular surfaces may modify the course of an inflammatory process. In tuberculous arthritis, for instance, the articular cartilage is destroyed first along the free surfaces where tuberculous granulations can grow and remove it; points of contact and pressure are protected from this invasion. In pyogenic arthritis, on the other hand, the cartilage is killed first at the point of contact and pressure, and proteolytic enzymes derived mostly from polymorphonuclear leucocytes assist greatly in the rapid removal of necrotic cartilage (30). The organism initiating the inflammatory process and the type of exudate it incites become subsidiary factors which also influence the rate and the progress of joint destruction. The irritated sensory nerve endings in joint structures may incite pain, cause reflex muscular spasm, and arrest motion; disturbances of both afferent and efferent limbs of the reflex arc, concerned with the conveyance of sensations on the one hand and motor impulses on the other, may variously result in lack of co-ordination, muscular imbalance, contractures, subluxations, and even complete breakdown of the architectural framework of the joint. The function of the joint as an organ of support and passive motion is interfered with in inflammatory, mechanical, circulatory, and neuromuscular disturbances.

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CONGENITAL ATRESIA OF THE EXTRAHEPATIC BILE DUCTS

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THE fact that more than 200 cases of congenital atresia of the extrahepatic bile ducts have been reported in the literature testifies that it is not a rare disease. Nearly every pediatrician sees one or more such cases during his active career. In spite of its rather frequent occurrence, nearly everyone has entertained a pessimistic attitude toward the outcome of this malady. It required the careful studies of Holmes, Ladd, and Donovan before a note of optimism could be sounded. These workers found that from 16 to 30 per cent of all cases of congenital atresia of the bile ducts were amenable to corrective surgery, and they made several valuable contributions concerning its diagnostic and therapeutic management.

During the past 5 years we have had an opportunity to study 5 cases of congenital atresia affecting the biliary radicals and have devised a method of roentgenographically visualizing the abnormal biliary tract at the time of operation, thus giving the surgeon an accurate visual concept of the problem which confronts him.

ETIOLOGY

Unfortunately, the pathogenicity of congenital atresia of the bile ducts is enshrouded in doubt. The most popular concept postulates that the atresia results from embryological malformations. In support of this view, Yllpo and Ladd point out that the bile ducts are tubular at first, and then by a proliferation of their epithelium, they are converted into solid cords. If normal development ensues, these cords undergo segmental vacuolation; the vacuoles coalesce and reestablish the ductal continuity. Partial or complete failure of vacuolation or fusion readily accounts for the great variety of anatomical anomalies.

Another theory holds that the atresia results from a primary intra-uterine hepatic cirrhosis, followed by a descending cholangitis which produces a fibrous occlusion of the biliary radicals. Rolleston and Hayne believe that toxins, either of maternal or fetal origin, initiate the cirrhosis. Accumulated evidence, however, suggests that the cirrhosis is the effect, rather than the cause, of the occlusion of the extrahepatic bile ducts. Some thought that a luetic hepatitis was the primary disease, but Holmes exploded this postulate by negative serological studies in all his cases.

It seems reasonable to believe, as pointed out by Holmes, that both of the theories mentioned have a certain degree of merit. The congenital malformation of the biliary radicals produces a stasis of bile, and the chemical irritation and the secondary cholangitis accelerates the fibrosing processes.

ANATOMY

Every conceivable type of atresic malformation of the bile ducts has been reported, but in spite of the multiform variations certain general groupings can be made: (1) agenesia of the extrahepatic bile ducts; (2) complete or segmental atresia of the hepatic ducts; (3) complete or segmental atresia of the choledochus; (4) complete or segmental atresia of the gall bladder and cystic duct with patency of the common and hepatic ducts; (5) complete or segmental atresia of the cystic and hepatic ducts—the type most frequently encountered; (6) atresia of the extrahepatic bile ducts with patency of the cystic duct and gall bladder; (7) agenesia of the extrahepatic ducts with the gall bladder connected directly to the duodenum; (8) mechanical obstruction of the patent bile ducts by plugs of mucus, inspissated plugs of bile, stones, and inflammatory edema of the ampulla Vater.

Holmes found that in 16 per cent of all atresic lesions, the cystic and hepatic ducts were patent. This is very significant for such deformities are amenable to surgical correction. Figure 1 represents the malformations encountered in our cases, none of which were of the remedial variety due to the complete obliteration of the common hepatic bile duct.

SYMPTOMS

Jaundice is the most constant symptom in congenital atresia of the bile ducts. There can be no doubt, however, that stenosing lesions of the gall bladder and cystic duct occur rather frequently, but as they do not impede the outward flow of bile they remain asymptomatic. The jaundice may be present at birth, but it usually appears during the first 3 weeks of life. Yllpo maintains that the biliary pigments are not elaborated in very large quantities until birth; therefore, the icterus does not become evident until hepatic saturation has been reached. The skin, sclera, and mucous membranes assume a yellowish-green color which differs from the yellowish tinge seen when biliary stasis occurs in the adult. The icterus is progressive but may show slight daily variations. The van den Bergh test gives a positive direct reaction and is not biphasic.

The stools are acholic, but at times they may give a positive reaction for bile. This does not indicate a patency of the biliary tract as is often thought but is caused by the cholemic blood oozing from the vessels of the intestinal mucosa. Large amounts of undigested fats may be recovered from the feces, particularly if the obstructive process occludes the ducts of Wirsung and Santorini. In spite of the dysfunctions of the liver and pancreas, these infants maintain a surprisingly good state of nutrition until toward the end.

The liver is always enlarged, irregular in outline, and may occupy most of the abdominal cavity. Splenomegaly and ascites usually result from passive congestion.

Petechial spots appear in the skin and mucous membranes while spontaneous hemorrhages occur from the nose, umbilicus, and gastro-intestinal tract. Profuse bleeding may follow the most trivial trauma. The bleeding and clotting time are usually normal or just slightly prolonged. The fragility of the red blood corpuscles is not altered, and the number of erythroblasts is not increased. If an anemia exists, it is of the secondary type.

DIAGNOSIS

Difficulty is frequently encountered in making an accurate diagnosis of congenital atresia of the bile ducts during the first month of life, but thereafter it can be accomplished with ease.

Icterus neonatorum occurs in 30 per cent of all newborn children. The jaundice is usually mild, appearing on the second or third day and soon subsides spontaneously. Even in severe cases the liver does not become enlarged, and the stools contain bile pigments.

Congenital hemolytic icterus seldom affects young children and can always be recognized by the increased fragility of the erythrocytes.

Luetic hepatitis may produce a mild jaundice but accurate maternal and infantile serological studies supplemented by the roentgenological examination of the long bones of the child afford accurate differentiation.

Erythroblastosis or icterus gravis affects the infant during the first few days of life and may be recognized by an increase in the number of erythroblasts in the circulating blood. The van den Bergh reaction is biphasic. Unfortunately, this disease runs a very rapid course and has an extremely high mortality; therefore, it seldom produces a chronic icterus.

Hemolytic icterus due to sepsis. Any infection which produces a rapid hemolysis of the red blood cells may initiate a mild icterus. The leucocytosis, the febrile reaction, the recognition of the infective focus, the presence of bile pigments in the stools, and the absence of hepatic enlargement usually lead to a correct diagnosis.

PROGNOSIS

Complete biliary stasis is incompatible with life. Surgical correction of existing abnormalities affords the only hope of relief. Refinements in diagnostic and surgical technique make it possible to salvage 20 to 30 per cent of these unfortunate children.

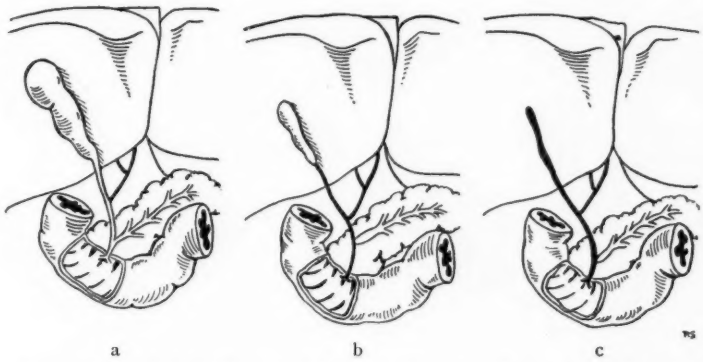

Fig. 1. Anatomical classification of the authors' cases. a, Cases 1 and 2. Complete atresia of the common hepatic and the right and left hepatic ducts. b, Cases 4 and 5. Complete atresia of the cystic duct, the common duct, and all hepatic bile ducts. Note the obstruction of the pancreatic ducts. c, Case 3. Complete atresia of the entire extrahepatic biliary system.

TREATMENT

Medical therapy has but one aim, that of improving the general condition of the infant so as to minimize the operative hazards. The administration of bile salts and vitamin concentrates helps compensate for existing deficiencies. Vitamin K, transfusion of whole blood, and hypertonic solutions of glucose are valuable in combating hemorrhagic tendencies.

Ladd and Donovan maintain that surgical intervention should be postponed until after the first month as positive diagnosis cannot be made prior to that time. Procrastination beyond this period, however, merely permits malnutrition, hemorrhages, and infectious processes to deplete the patient's reserve and thus increase the operative risk.

Surgical correction of the biliary stasis is predicated on two fundamental premises; first, the recognition of existing abnormalities, and second, the ability to provide a new channel through which the bile may flow into the gastro-intestinal tract. All too frequently, neither of these objectives can be accomplished, and the patient dies.

The multiplicity of variations, the small caliber of the infantile bile ducts, the enlargement of the liver, and the associated periductal fibrosis incident to concomitant infections certainly do not facilitate the recognition of the congenital deformities. Anyone who has attempted the tedious task of dissecting out the improperly developed gall bladder and bile ducts appreciates the futility of such procedures. One soon becomes lost in the maze of blood vessels, nerves, bile stained connective tissue, and fibrotic bile ducts. This is particularly true if the hepatic ducts are impervious, for then the distal segments of the biliary tract are small, collapsed, and undeveloped. Ladd appreciated this difficulty and suggested that the collapsed gall bladder be distended with saline solution so that bile ducts could be more readily identified. This procedure is invaluable if all the bile ducts are patent but if the cystic duct is obliterated, the saline solution can not distend the choledochus and hence is of no value.

Previously we had demonstrated that it was possible, by means of contrast roentgenography (1, 5), to obtain exact anatomical patterns of the adult biliary tract while the patient was on the operating table. It was hoped that these cholangiographic studies might prove helpful in visualizing and locating the obstructive lesions affecting the diminutive biliary tract of the infant. These cholangiograms not only facilitated the classification of the obstructions but greatly aided the surgeons in selecting the proper remedial procedure.

CASE HISTORIES

Case 1. (Hicken.) M. E., a white male child, 5 months of age, was referred by Dr. Bruce Eldridge,

because of a progressive, painless jaundice. The icterus was first observed 3 weeks following birth and had steadily increased in intensity. Several times it was "thought that the stools contained bile." Malnutrition, asthenia, hemorrhages from the bowels and susceptibility to pulmonary infections finally convinced the parents that surgical intervention was imperative.

On examination the liver was found to occupy the major portion of the abdominal cavity. It was firm, irregular, and tender. There was mild ascites. The spleen was about twice the normal size. There were 3,500,000 erythrocytes, 12,800 leucocytes, and the hemoglobin was 70 per cent. The bleeding and clotting time, as well as the fragility of the red blood corpuscles, were all of normal values. The Kline and Wassermann reactions were negative.

Vitamin concentrates, bile salts, hypertonic solutions of glucose, and transfusions of whole blood were given in an effort to combat the malnutrition. An exploratory laparotomy was performed, but the gall bladder and extrahepatic bile ducts could not be identified in the periportal structures. It was thought that the gall bladder might be collapsed, therefore a needle was inserted into the tissues occupying the gall bladder fossa and 1 cubic centimeter of contrast substance was slowly introduced (Fig. 2). Much to our surprise, the small atrophic gall bladder became distended and could be readily palpated. Cholangiograms demonstrated the gall bladder, the cystic and common bile ducts, and the pancreatic ducts to be patent. The contrast substance readily entered the duodenum (Fig. 3). The visualizing media, however, was unable to enter the impervious hepatic ducts because of an occluding fibrosis. Further exploration revealed that both the right and left hepatic ducts had been converted into fibrous cords and that the obstructive process was intrahepatic. It was obvious that there was no possible way in which the liver could be decompressed and the bile diverted into the duodenum; hence, the abdomen was closed. A terminal bronchopneumonia proved fatal 47 hours later.

Necropsy examination demonstrated that the cholangiographic studies were correct. The gall bladder, cystic, and common bile ducts were normal, but the common hepatic, the right and left hepatic ducts, and the intrahepatic ducts were solid cords. Histologic studies failed to demonstrate either the duct lumen or its mucosal lining. The fibrosing process followed each of the hepatic ducts into the liver substance for a distance of 4 centimeters, thus making decompressive procedures impossible. The intrahepatic bile ducts were definitely dilated, and the liver presented the picture of bile stasis and cirrhosis.

Case 2. (Hicken.) H. V., a white male child, 3 months of age, entered the University of Nebraska Hospital on the pediatric service of Dr. E. W. Bantin. The icterus was present at birth and had progressively increased in severity. The stools were always acholic. Purpuric spots had been observed on the knees and ankles at various times. A pernicious vomiting and a persistent bloody diarrhea had rapidly depleted the physical reserve.

The liver was hard, irregular, and extended downward to the crest of the ilium.The spleen was about 2½ times larger than it should be. The icteric index was 60 and the van den Bergh gave a direct and positive reaction. The urine contained bile but the stools were acholic. The Kline and Wassermann reactions were negative. There were 3,000,000 red blood cells, 11,000 leucocytes, 110,000 blood platelets, and 3.5 per cent reticulocytes. The fragility tests were normal.

An exploratory laparotomy revealed a large bile-stained cirrhotic liver. The gall-bladder fossa contained a small amount of smooth tissue from which no fluid could be aspirated. In spite of a most meticulous dissection we were unable to identify the common bile duct. Attempts to distend the collapsed gall-bladder with saline solution, as advised by Ladd, failed. In hopes of visualizing the bile ducts the duodenum was opened and a small cannula was inserted into the ampulla of Vater through which 1 cubic centimeter of contrast substance was introduced. Cholangiograms demonstrated the ampulla of Vater, choledochus, pancreatic ducts, cystic duct, and gall bladder to be normally developed and patulous. The contrast media, however, failed to enter the common hepatic duct thus demonstrating a complete obliteration of its lumen. No decompressive procedures could be employed as the biliary stasis resulted from occlusion of the intrahepatic segments of the right and left hepatic ducts. The abdomen was closed, and the patient succumbed 36 hours later.

Necropsy studies revealed the cirrhotic liver to weigh 300 grams, and the spleen was twice its normal size. Pressure on the small gall bladder forced the contrast media, which had been introduced at the time of operation, along the bile ducts out into the duodenum, thus attesting to the patency of these radicals. The hepatic ducts could not be identified for even microscopic sections through the periportal fibrotic tissues failed to locate mucosal tissues or ductal structures. Like Case 1, this must be classified as complete atresia of the common hepatic and the right and left hepatic ducts.

This case emphasizes the point that the visualizing media may be introduced into any portion of the extrahepatic biliary system that is patent. Whenever possible, it should be injected into the gall bladder, but if this cannot be accomplished, the exploring needle may be inserted into the choledochus, the hepatic ducts, or even into the ampulla of Vater. It is true that these transduodenal injections carry a high mortality but when one appreciates the seriousness of these conditions, and when all other methods have failed to

locate the obstructive lesion, then one is justified in performing these transduodenal cholangiograms.

CASE 3. (Best-Hicken.) B. J., a white female child, 5 months of age, was first seen with Dr. R. R. Best. A progressive, painless jaundice since birth, combined with acholic stools, enlargement of the liver and spleen, and a normal fragility test, suggested the diagnosis of congenital atresia of the bile ducts.

We explored the abdomen and found the liver to be greatly enlarged. The dense mass of bile stained cicatricial tissue which occupied the area surrounding the portal vein made it impossible to identify any portion of the extrahepatic biliary system. The gall bladder fossa was very small and no remnants of the gall bladder could be detected. In spite of a most tedious and painstaking dissection we were unable to identify any portion of the bile ducts. The impervious state of the extrahepatic ducts made it impossible to utilize the visualizing cholangiograms; therefore we were compelled to make the diagnosis of complete atresia of the entire extrahepatic biliary system. Autopsy studies confirmed the diagnosis and also revealed the futility of attempting decompressive procedures.

CASE 4 (Henske.) P. P., a white male child was first seen by Dr. Henske when only 3 months of age.

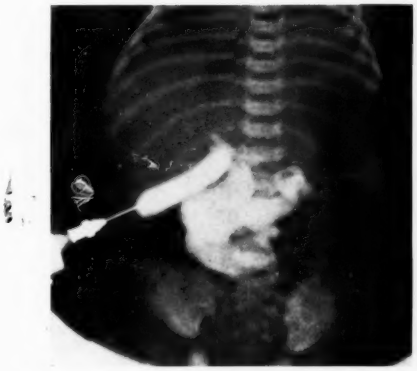

Fig. 3. The cholangiogram clearly outlines the gall bladder, common bile duct, and duodenum, indicating that they are patent. An obstructive lesion of the common hepatic bile duct prevented the contrast substance from outlining the right and left hepatic ducts or the intrahepatic radicals.

At this time, the child had a pronounced jaundice, acholic stools, biliuria, and enlargement of the liver. The diagnosis of congenital atresia of the bile ducts was made and operation was advised. The parents declined permission for operation; therefore the

Fig. 2. Demonstrating the technique of introducing the contrast substances into the gall bladder in order to obtain accurate roentgenographic patterns of the bile ducts during the operation.

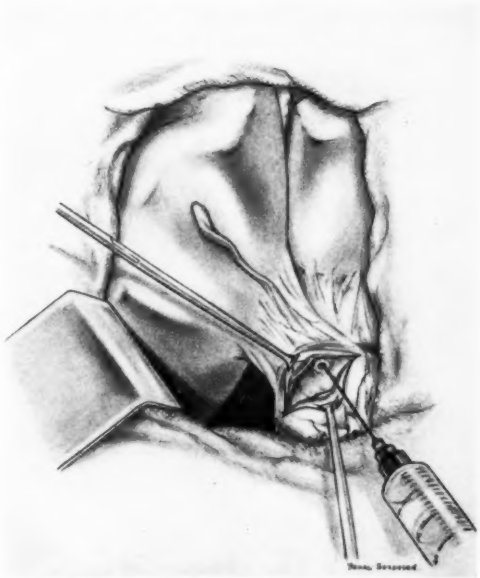

Fig. 4. When the extrahepatic bile ducts cannot be identified, we have opened the duodenum and informative cholangiograms have been obtained by introducing the contrast substance directly into the ampulla of Vater.

Fig. 5a.

Fig. 5b.

Fig. 5. a, The cholangiogram of Case 2 clearly outlines the ampulla of Vater, pancreatic ducts, choledochus, and the gall bladder. However, the failure of contrast substance to enter the hepatic ducts and intrahepatic radicals indicates that the obstructive lesion involves these segments. b, Gross specimen showing small gall bladder and patent cystic and common bile ducts. The common hepatic, the right and left hepatic ducts, and intrahepatic segments were completely occluded.

youngster was discharged. In spite of high caloric diets rich in vitaminic and bile-salts content, the youngster died 3 weeks later from a severe pneumonitis and gastro-intestinal hemorrhages.

Necropsy examination confirmed the hopeless nature of this disease by showing the gall bladder to be patent and slightly distended with mucus. The cystic duct, hepatics, and choledochus had been converted into fibrous cords. No plastic operation could have been employed.

CASE 5 (Henske.) L.C., a white female child, was apparently normal at birth but 3 days later developed a slight icterus. The jaundice slowly increased in intensity and failed to respond to the common remedial agents so the patient was referred to Dr. J. A. Henske. Biliuria, acholic stools, intense jaundice, malnutrition, melena, and hepatomegaly suggested the diagnosis of congenital obstruction of the bile ducts.

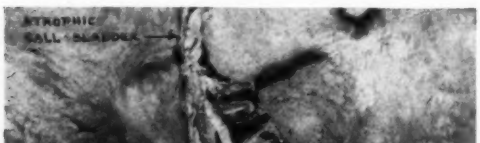

Fig. 6. Small atrophic gall bladder with complete atresia of all other components of the intrahepatic and extrahepatic bile ducts.

Dr. Henske did not believe that this case was remedial to surgery; therefore he gave supportive treatments in the form of vitamin concentrates, blood transfusions, and bile salts. During the next 3 weeks she made a pronounced improvement but later contracted a pneumonitis and died when 5 months old.

At autopsy, Dr. C. P. Baker found the abdominal cavity to contain 300 cubic centimeters of bile stained fluid. The liver weighed 400 grams and was deeply pigmented and cirrhotic. Its markings were indistinct, but the intrahepatic ducts were all dilated. The gall bladder was small and atrophic but patulous. The cystic duct, the hepatic ducts, and the choledochus were all fibrotic and occluded. The ampulla of Vater and the pancreatic ducts were all normal. It was evident that surgical correction of obstructive lesions was absolutely impossible.

SELECTION OF OPERATION

Not all types of atresic obstructions are amenable to surgery for some of them are so extensive that complete restoration is impossible. In other varieties the lesion is situated in such inaccessible places that new conduits for the passage of bile cannot be constructed. Those atresic lesions which can be corrected are represented by the pictograms seen in Figure 7. It will be noted that in every instance the intrahepatic radicals, the right and left hepatics, and the common

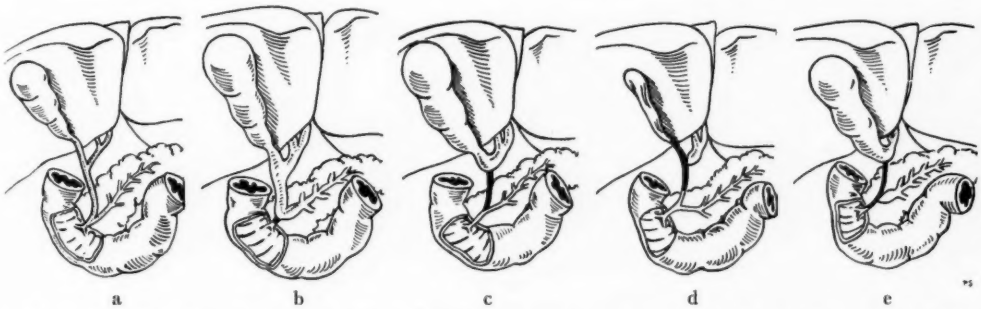

Fig. 7. Remedial lesions of the bile ducts. a, Normal biliary system. b, Obstruction of the ampulla of Vater may be relieved by flushing, dilatation, cholecysto-enterostomy, and choledocho-enterostomy. c, Occlusion of the upper two-thirds of the choledochus relieved by cholecysto-enterostomy. d, Occlusion of the upper two-thirds of the choledochus and the cystic duct relieved by hepatico-duodenostomy. e, The obstruction of the bile ducts can be overcome by a cholecysto-enterostomy. A pancreatico-duodenostomy could be attempted later.

hepatic bile ducts were all patent. The bile must have free access to the extrahepatic ducts, and there must be sufficient amount of normal ductal tissue to permit short-circuiting plastic operations. The type of operation employed depends entirely on the extent and location of the occlusion.

If the obstructive lesion is located in the ampulla of Vater (Fig. 7 b), distal to the openings of the pancreatic ducts, the outlook is very favorable. Ladd has pointed out that if plugs of inspissated bile or mucus should occlude the choledochoduodenal orifice that they can be dislodged by gently irrigating the gall bladder with a solution of saline. If this fails to disengage the impacted plugs, he suggests that a small probe be passed along the choledochus into the duodenum. If such simple procedures relieve the obstruction nothing more need be done. In some cases, however, there is a dense fibrous occlusion of the ampullar orifice making it necessary to perform a choledochoduodenostomy. This is the operation of choice but if the patient's condition is serious, a simple cholecystogastrostomy may suffice. In all such operations it is essential to know that there is no obstruction proximal to the site of anastomosis. Such information is readily obtained by means of cholangiography.

Occasionally a segmental atresia involving the upper two thirds of the choledochus will be encountered (Fig. 7 c). If the ampullic segment is patent, thus affording an outlet for the pancreatic hormones, a cholecysto-enterostomy is very effective. One must be certain that the cystic duct is patent, otherwise the liver will not be decompressed of its bile stasis.

The combined atresia of the choledochus and the cystic duct is frequently encountered (Fig. 7 d). The gall bladder may be collapsed or distended with a yellowish mucoid material which may be mistaken for bile. The introduction of contrast media into the gall bladder will readily visualize the occlusive lesion of the cystic duct and thus prevent a useless cholecysto-enterostomy. When the common hepatic duct is distended, it may be anastomosed to the duodenum. Ladd, with inexhaustible ingenuity, performed such an operation by employing a small rubber catheter which he inserted into the hepatic duct and then anchored to the duodenum, thus forming a new channel for the flow of bile. The minuteness of the infantile ducts makes this a most delicate procedure but one worth attempting.

Obliteration of the lower third of the common bile duct is a serious condition (Fig. 7 e) for it means that the ducts of Wirsung and Santorini are definitely occluded. It is true that a cholecysto-enterostomy will completely relieve the biliary stasis but unless a new outlet is provided for the pancreatic secretions the ensuing asthenia may prove fatal. By means of cholangiographic studies one can determine the status of the pancreatic ducts and, while plastic repairs may be difficult to perform, it is a challenge which must be met.

It is evident that the intrahepatic radicals and the common hepatic bile duct with its two tributaries must be patent if a reconstructive decompression of the liver is to be accomplished. If a new conduit for the egress of bile can be established the patients make a complete and gratifying recovery. Many cases that have been reported as inoperable were so classified because of the surgeon's inability to isolate the biliary radicals and not because of the type of atresic lesion. These cholangiograms not only visualize the exact location and extent of the existing malformations but also furnish evidence which is helpful in selecting the proper therapeutic procedure. This is clearly portrayed in Cases 1 and 2 as the cholangiograms revealed the gall bladder, cystic duct, and choledochus to be patulous, hence a cholecysto-enterostomy seemed indicated, but they also demonstrated a complete atresia of the common hepatic ducts and its radicals, thus rendering such an operation ineffective.

Attempts to decompress the biliary stasis by scarifying the surface of the liver in such a manner that the peripheral intrahepatic bile ducts would be severed and then anastomosing this traumatized area to the stomach have, in our hands, resulted in death either from hemorrhage or infections.

SUMMARY

1. Embryological malformations apparently account for the congenital atresias and obstructions of the extrahepatic bile ducts.

2. This disease is usually characterized by a progressive, painless jaundice which appears during the first 3 weeks of life and is associated with hemorrhagic tendencies, malnutrition, enlargement of the liver, splenomegalia and ascites.

3. Corrective surgery, which establishes a new channel for the flow of bile into the intestinal tract, thus decompressing the liver, offers the only hope of cure. It has been held that all of these cases are hopeless but recent studies indicate that from 20 to 30 per cent are amenable to surgery.

4. The method of visualizing these ductal deformities at the time of operation is presented. These cholangiographic studies determine the location and extent of the obstructive lesion.

5. Five cases of congenital atresia of the bile ducts are presented but in all of them, the obstructive lesions were in such positions that it precluded the possibility of surgical repair.

We express our appreciation to Dr. R. R. Best, Dr. J. A. Henske, Dr. E. W. Bantin, and to the University of Nebraska College of Medicine, Omaha, Nebraska, for their generous assistance in the preparation of this manuscript.

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THE SUPERIORITY OF NEOARSPHENAMINE AND SULFATHIAZOL IN THE THERAPY OF STAPHYLOCOCCUS AUREUS INFECTIONS IN MARROW CULTURES

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THE development of the marrow culture method (15) has made possible accurate quantitative comparison under controlled conditions of the relative effectiveness of therapeutic agents against bacterial infections in the presence of living human cells (1, 9 to 14). The Staphylococcus aureus is a frequent cause of human infection, and is relatively resistant to therapy with sulfanilamide and sulfapyridine (8). LeCocq (5) and LeCocq (6) have obtained good results in Staphylococcus aureus infections with neoarsphenamine. The new compounds, sulfathiazol and sulfamethylthiazol, have been reported (2, 3, 4, 7) to be more effective in Staphylococcus aureus infections than other drugs. The object of this study was to determine the most effective of these drugs in the therapy of Staphylococcus aureus infections and the ideal concentration.

METHOD

Cultures of human bone marrow were prepared as described (1, 9–15). To about 50 cubic centimeters of culture containing about 100,000,000 nucleated marrow cells in one vial was added with syringe and needle a dilution of a culture of Staphylococcus aureus in Hartley broth. After thorough mixing, pour plates were made for colony counts, and equal volumes of about 8 cubic centimeters were then transferred to each of a number of 30 cubic centimeter vaccine vials. This insured that each vial contained the same number of the same strain of organisms and the same number of cells in identical medium. To each of these, except the control, enough of the drug to be studied was added to give the desired concentrations. The cultures were then placed in incubator at 37 degrees C., and pour plate colony counts and smears were made at intervals. This technique insures that the only variable is the presence of the drug.

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RESULTS

In all, a total of 20 experiments similar to the 7 shown in Tables I to VII were done. They were consistent in showing that sulfanilamide and sulfapyridine were relatively ineffective (Tables I to VII, and Figures 17, 18, 19, and 21 in reference 12) and that neoarsphenamine was very effective (Tables I to VIII). Preliminary reports of these studies were made (12, 13). It was soon discovered that neoarsphenamine in concentrations above 1:70,000 not only led to sterility of the cultures but also destroyed the marrow cells, and that in concentrations between 1:70,000 and 1:150,000 the marrow cells were damaged. Concentrations below 1:400,000 were much less effective than higher concentrations (Table I), and concentrations below 1:200,000 were somewhat less effective. The ideal concentration, therefore, is apparently 1:150,000, or about 6 parts per million. In this concentration, however, the drug has to be present for 6 to 48 hours or longer to be effective

TABLE I.—COMPARATIVE EFFECTIVENESS OF SULFANILAMIDE, SULFAPYRIDINE AND VARYING CONCENTRATIONS OF NEOARSPHENAMINE ON STRAIN 1, TABLE VIII.

Hours	0	24	72
Control	60	87,500,000	800,000,000
Sulfanilamide, 1:10,000	60	38,000,000	250,000,000
Sulfapyridine, 1:20,000	60	12,000,000	162,000,000
Neoarsphenamine, 1:200,000	60	50	2,700
Neoarsphenamine, 1:263,000	60	600	5,000,000
Neoarsphenamine, 1:400,000	60	4,000	13,000,000

TABLE II.—COMPARATIVE EFFECTIVENESS OF VARYING CONCENTRATIONS OF NEOARSPHENAMINE ON STRAIN 1, TABLE VIII.

Hours	0	3	6	24	48
Control	10	30	375	400,000,000	
Neoarsphenamine, 1:67,000	10	10	5	0	0
Neoarsphenamine, 1:80,000	10	5	0	0	0
Neoarsphenamine, 1:125,000	10	20	0	0	0
Neoarsphenamine, 1:200,000	10	20	25	25	12

TABLE III.—COMPARATIVE EFFECTIVENESS OF VARYING CONCENTRATIONS OF NEOARSPHENAMINE ON STRAIN 1, TABLE VIII.

Hours	0	5½	27	72
Control	50	870	>400,000,000	
Neoarsphenamine, 1:83,300	50	16	0	0
Neoarsphenamine, 1:97,300	50	25	4	0
Neoarsphenamine, 1:130,000	50	150	100	5
Neoarsphenamine, 1:156,000	50	40	5	2
Neoarsphenamine, 1:200,000	50	40	25	1

(Tables I to VII). Sulfathiazol and sulfamethylthiazol in concentrations of 1:10,000 (Tables IV to VII) were also very effective, but were not as effective as neoarsphenamine (Tables IV to VIII, and Figures 1–4). The results of all the tests made on each of the strains of Staphylococcus aureus investigated are summarized in Table VIII. Note that in 1:10,000 concentrations sulfanilamide was ineffective, and sulfapyridine was only slightly effective in both tests reported, as was expected from our previous studies. Sulfathiazol in 1:10,000 concentrations was definitely superior to sulfapyridine. In the 10 experiments in which it was studied sulfathiazol was very effective in 2, effective in 2, slightly effective in 5, and ineffective in only 1. It failed to result in sterility of any culture, although if smaller inoculations had been made it is possible that it might have produced sterility. Sulfamethylthiazol in 1:10,000 concentrations was almost as effective as sulfathiazol in the marrow cultures. In the

TABLE IV.—COMPARATIVE EFFECTIVENESS OF SULFATHIAZOL, SULFAMETHYLTHIAZOL AND NEOARSPHENAMINE ON STRAIN 3, TABLE VIII.

Hours	0	2	4	24	48
Control	2,300	3,600	10,000	>400,000,000	
Sulfathiazol, 1:10,000	2,300	3,200	13,500	950,000	>400,000,000
Sulfamethylthiazol, 1:10,000	2,300	2,400	11,000	1,400,000	>400,000,000
Neoarsphenamine, 1:150,000	2,300	2,600	2,500	28,000	32,900,000

TABLE V.—COMPARATIVE EFFECTIVENESS OF SULFATHIAZOL, SULFAMETHYLTHIAZOL AND NEOARSPHENAMINE ON STRAIN 4, TABLE VIII.

Hours	0	24	28	46	48
Control	50,000?	>400,000,000			
Sulfathiazol, 1:10,000	50,000?	1,300,000	1,600,000	>400,000,000	
Sulfamethylthiazol, 1:10,000	50,000?	2,000,000	2,200,000	>400,000,000	
Neoarsphenamine, 1:150,000	50,000?	10,000	6,800		1,700

TABLE VI.—COMPARATIVE EFFECTIVENESS OF SULFATHIAZOL, SULFAMETHYLTHIAZOL AND NEOARSPHENAMINE ON STRAIN 5, TABLE VIII.

Hours	0	4	24	28	46	48
Control	11,100	162,000	>400,000,000			
Sulfathiazol, 1:10,000	11,100	60,000	1,000,000	1,500,000	>400,000,000	
Sulfamethylthiazol, 1:10,000	11,100	100,000	1,400,000	1,000,000	>400,000,000	
Neoarsphenamine, 1:150,000	11,100	11,800	1,200	1,600		7,000

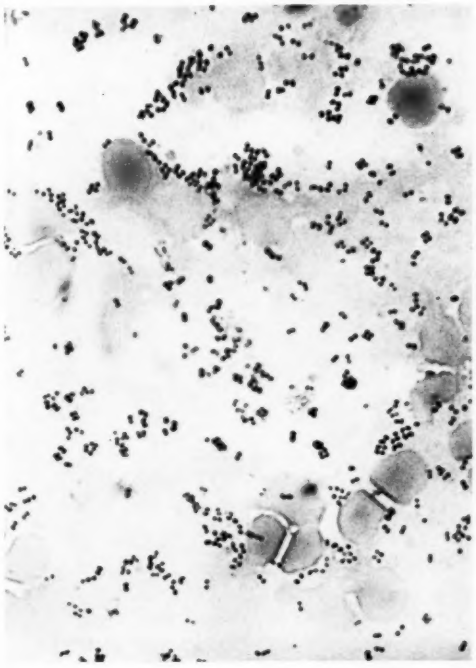

Fig. 1. Photomicrograph of the control from the experiment which is shown in Table VI at 24 hours. Wright's stain, ×810.

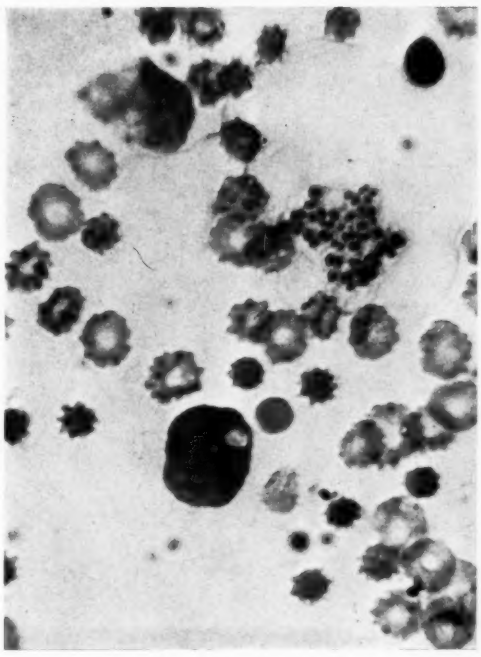

Fig. 2. Photomicrograph of the culture containing 1:10,000 sulfathiazol at 24 hours from the experiment shown in Table VI. Wright's stain, ×810.

7 studies, it was very effective in 1, effective in 1, slightly effective in 4, and ineffective in 1. Neoarsphenamine in concentrations above 1:100,000 resulted in sterility of the cultures in all 13 trials, but damaged or destroyed marrow cells in these concentrations. In concentrations of 1:100,000 to 1:200,000 neoarsphenamine was superior to any other therapy investigated. Eight of 29 cultures investigated were sterilized, in 16 cultures it was very effective, in 3 it was effective, in 3 it was slightly effective, and in only 1 was it ineffective. In concentrations of 1:200,000 to 1:400,000 neoarsphenamine was of some value but not as effective as in higher concentrations. In 8 studies 2 cultures became sterile, in 3 it was very effective, in 2 it was effective, and in 1 it was slightly effective. It is apparent from this study that in marrow cultures the ideal concentration of neoarsphenamine is 1:150,000 to 1:200,000.

ANALYSIS OF STUDY

These studies indicate that, as with the Streptococcus viridans (12, 13, 14), sulfanilamide is ineffective against Staphylococcus aureus in concentrations obtainable in the blood stream. Sulfapyridine is somewhat superior to sulfanilamide, but it is much less effective than sulfathiazol or sulfamethylthiazol. Sulfathiazol and sulfamethylthiazol are very effective, but they rarely lead to sterility in marrow cultures. In 1:10,000 concentrations they are definitely inferior to neoarsphenamine in concentrations of 1:100,000 to 1:200,000.

The development of an accurate method for blood arsenic by Raulston, Magnuson and Chaney (16, 17) made possible the calculation of the laws governing the distribution of the drug in the body and its elimination from the blood in man. After a single dose the drug is distributed evenly through the blood volume,

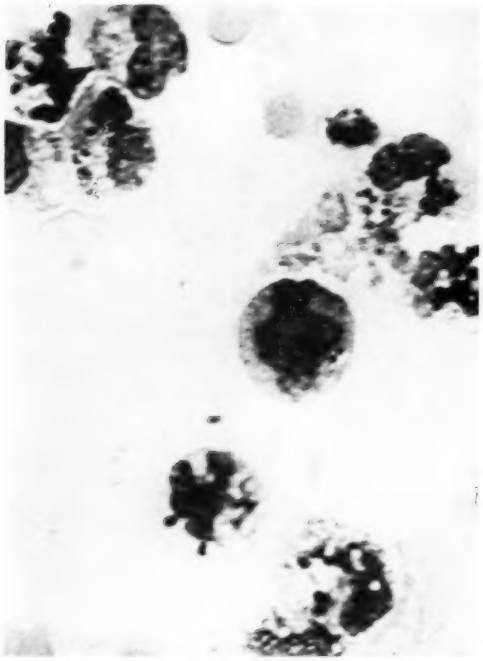

Fig. 3. Photomicrograph of the culture containing 1:10,000 sulfamethylthiazol at 24 hours from the experiment shown in Table VI. Wright's stain, ×810.

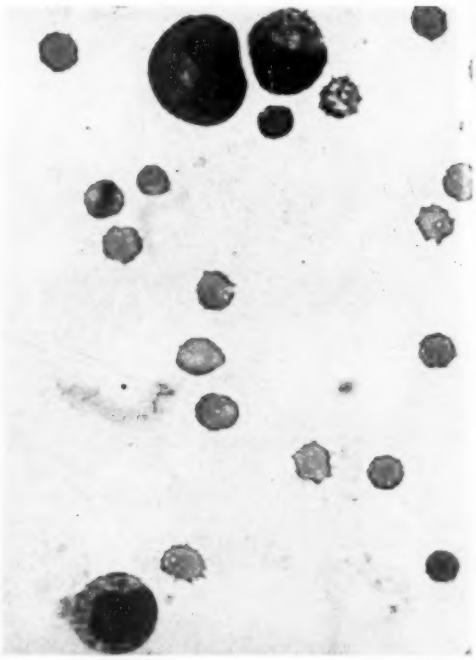

Fig. 4. Photomicrograph of the culture containing 1:150,000 neoarsphenamine at 24 hours from experiment shown in Table VI. Wright's stain, ×810.

but rapidly leaves the blood and after 2 to 3 hours is distributed almost evenly throughout the body tissues. From this time on the blood level drops comparatively slowly. During both periods the drug leaves the blood by a clearance mechanism which corresponds to a clearance of about 50 cubic centimeters of blood per hour. To maintain continuously the blood level desired of one part in one hundred and fifty thousand, or 200 gamma of arsenic per 100 cubic centimeters, as the results are reported, one should give $\frac{1}{150,000}$ of the body weight in grams of neoarsphenamine $\left(\text{or}\frac{\text{body weight in kg.}}{150}=\frac{\text{body weight in pounds}}{330}\right)$ the first day, divided into 3 or 4 spaced doses. Each subsequent day until 6 to 10 days after the patient is afebrile give three-fourths of the total dose given on the first day, divided

TABLE VII.—COMPARATIVE EFFECTIVENESS OF SULFANILAMIDE, SULFAPYRIDINE, SULFATHIAZOL, SULFAMETHYLTHIAZOL AND NEOARSPHENAMINE ON STRAIN 7, TABLE VIII.

Hours	0	4	8	24½	74
Control	1,100	20,400	260,000	450,000,000	
Sulfanilamide, 1:10,000	1,100	10,000	120,000	120,000,000	
Sulfapyridine, 1:10,000	1,100	8,400	28,700	1,500,000	>400,000,000
Sulfathiazol, 1:10,000	1,100	6,800	9,300	34,000	>400,000,000
Sulfamethylthiazol, 1:10,000	1,100	7,200	18,000	120,000	>400,000,000
Neoarsphenamine, 1:250,000	1,100	157	1,250	65	

TABLE VIII.—SUMMARY OF RESULTS WITH ALL DRUGS STUDIED ON SIX STRAINS OF STAPHYLOCOCCUS AUREUS.

Strains	1	2	3	4	5	6
Sulfanilamide	1 I					1 I
Sulfapyridine	1 S					1 S
Sulfathiazol			2 S 1 I	2 V 1 S	1 S	2 E 1 S
Sulfamethylthiazol			2 S 1 I	1 V 1 S	1 S	1 E
Neoarsphenamine, 1:100,000	13 O					
Neoarsphenamine, 1:100,000 to 1:200,000	8 O 10 V 2 E	2 V	1 E 1 S 1 I	1 V 2 S	1 V	2 V
Neoarsphenamine, 1:200,000	2 O 2 E 1 S	2 V				1 V

O—the culture was sterile when the colony count in the control was more than 400,000,000 per c.cm.
V—very effective, or the colony count ranged from 1 to 10,000 when the colony count in the control reached 400,000,000.
E—effective, or the colony count ranged from 10,000 to 200,000 when the colony count in the control reached 400,000,000.
S—slightly effective, or the colony count ranged from 200,000 to 60,000,000 when the colony count in the control reached 400,000,000.
I—ineffective, or the colony count was over 60,000,000 when the colony count in the control reached 400,000,000.

into three spaced doses. For example: If the body weight of the patient is 60 kilograms, give a total of 0.4 gram in divided doses of 0.1 gram each at 8 a.m., 11 a.m., 2 p.m., and 8 p.m. Every subsequent day give 0.1 gram at 8 a.m., 3 p.m., and 10 p.m. On the third day and subsequently at intervals, just before giving the 8 a.m. injection, take blood for blood arsenic determinations (17). Adjust the doses up or down as necessary to keep the blood arsenic level between 100 and 130 gamma per 100 cubic centimeters when the blood is taken at this time.

Sulfathiazol in doses sufficient to give a blood level of 8 to 10 milligrams per 100 cubic centimeters may be used alone in the milder staphylococcus infections, and in conjunction with neoarsphenamine in the most severe infections. Sulfamethylthiazol should not be used because of its tendency to produce polyneuritis. If difficulty is encountered in securing adequate blood levels of sulfathiazol, restriction of fluid may be more effective than merely increasing the dose.

Clinical studies are in progress and will be reported when enough data have been secured. Obviously much work remains to be done, both by the marrow culture technique and clinically, to determine whether any other arsenical is as effective as neoarsphenamine. This report is made in the hope that those who have facilities for carefully controlled clinical investigation will aid us in determining as soon as possible the clinical value of this treatment in serious staphylococcic infections, such as staphylococcic bacteriemias and meningitis.

SUMMARY

This series of controlled quantitative studies of the relative effectiveness of sulfanilamide, sulfapyridine, sulfathiazol, sulfamethylthiazol and neoarsphenamine against Staphylococcus aureus infections were made by the marrow culture method. In the marrow cultures neoarsphenamine in concentrations of 1:150,000 to 1:200,000 was the most effective preparation. In concentrations of 1:10,000 sulfathiazol and sulfamethylthiazol were of definite value but rarely led to complete sterility; sulfapyridine was only slightly effective; and sulfanilamide was practically ineffective. Concentrations of neoarsphenamine above 1:150,000 were toxic to the marrow cells, and concentrations above 1:70,000 killed all cells. These studies suggest that administration of neoarsphenamine in intermittent courses of repeated small doses together with sulfathiazol in doses sufficient to give a blood concentration of 1:10,000 is worthy of controlled clinical investigation in serious Staphylococcus aureus infections.

We are indebted to the Department of Medical Research of the Winthrop Chemical Company, Inc., for the neoarsphenamine and sulfamethylthiazol, to Merck & Co., Inc., for the sulfapyridine, and to the Squibb Institute for Medical Research for the sulfathiazol used.

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THE HYDRATION OF HYPERTHYROID PATIENTS AND ITS RELATIONSHIP TO EDEMA, ESPECIALLY CEREBRAL

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THE work of Coller, Maddock and their associates (6) has done much to focus attention on the subject of hydration of the surgical patient and as a result of their efforts, surgeons in general are "water-conscious" to an extent unthought of 10 years ago. This has been to the enormous benefit of surgical patients as a whole and has keenly stimulated interest in the derangement of physiological processes that occur in dehydration. Many of the problems of water balance in disease are unsolved, however, since our knowledge of colloidal chemistry is still fragmentary compared with what we may hope it to be in the future. That the figures Coller and Maddock consider a desirable water intake for the average surgical patient are ideal is not clear as yet. Theoretical objection to the application to disease of Newburgh's formulas, which involve a number of assumptions, have been voiced by Peters. The estimation of insensible perspiration, moreover, by the weighing of as many components as are involved in the calculations of Coller and Maddock introduces quantities of weight of much greater magnitude than the calculated weight of water lost by insensible perspiration. Very trifling errors in the former would therefore be greatly magnified in the latter. There is also a certain dissatisfaction among clinicians with the results of giving sufficient water, in whatever solution, and particularly by vicarious routes of intake, necessary to produce a urinary output of 1500 cubic centimeters per day plus an allowance of 1500 to 2000 cubic centimeters daily for insensible perspiration above all other fluid losses. Elderly patients in general are more apt to develop water retention on such a regimen than are younger subjects, even though they originally have not clinically demonstrable, functional or anatomical disorders of the circulatory system. The occurrence of pulmonary edema, which often overtakes the patient suddenly, in such postoperative patients may precipitate a critical situation.

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The author has been particularly interested in this phenomenon in patients with hyperthyroidism and it is with the problem of their hydration that we are immediately concerned. As stated, there is still much mystery attached to the ways in which the tissues handle water, particularly in disease, as studies of even the factors known to be involved are incomplete. A tendency to water retention, i.e., to edema, is determined primarily by variations in balance between hydrostatic pressure in the capillaries and the colloid osmotic pressure of the blood; the latter is affected by changes of the plasma proteins, particularly of the albumin fraction, and of the electrolytes. There is every reason to think that hydrostatic pressure in the capillaries is increased in hyperthyroid patients, increased minute output of the heart and high pulse pressure being well recognized; peripheral vasodilatation may well be a compensatory effort to keep capillary pressure as nearly normal as possible, as well as increasing dissipation of heat by favoring both radiation and insensible water loss from the skin. The figures of Coller and Maddock (5) indicate an increase both in percentage and amount of heat lost through vaporization of water with rising metabolism and the assumption seems currently reasonable that capillary dilatation in the lungs occurs also and serves the same ends.

A search for disturbances in the concentration of electrolytes (10) of the blood in thyrotoxicosis has been fruitless so far; this is hardly surprising, so far as the cations are concerned, for their constancy, particularly that of sodium, is guarded with the utmost care. Total electrolyte concentration and pattern seem to vary in disease much less widely than colloid osmotic pressure or even the total osmotic pressure of the plasma (12). There is evidence (1, 4) that the plasma proteins are decreased, especially the albumin fraction (1) and unpublished data of our own indicate a further rather sharp decrease of albumin in the immediately postoperative phase, often with a rise in globulin and a reversal of the albumin-globulin ratio. For any prediction of the tendency to water retention it is necessary to know the plasma volume as well as the concentration of plasma proteins, and there is no agreement as to the facts in hyperthyroidism. Rowntree and Bartels refer to an increased plasma volume while Brown and McCray, and Frazier and North consider it to be decreased. This point has an important bearing on the situation and needs clarification. It must be realized that the blood does not necessarily take part in edema and that with reduced serum protein the volume of circulatory blood is probably not large (12). Further factors modifying the behavior of water in the body are tissue tension and the existence of anoxemia. Large weight loss, which favors the development of decreased tissue tension, and a tendency to anoxemia are common features in the clinical course of hyperthyroidism; an "arteriolarization" of the venous blood, that is, a decreased A-V difference of oxygenation, has been demonstrated by Gladstone. One cannot simply count on the functional ability of the kidneys to excrete all water above the needs of the tissues in disease and this is a trap into which clinicians are prone to fall. The kidneys are more concerned with the maintenance of serum electrolytes in normal quantities and concentration than anything else; while they are not indifferent to the total body water content, their response seems more directly related to composition than to volume of body fluids (12).

The importance of the considerations outlined lies in the fact that the known or suspected deviations from normal mechanisms in hyperthyroidism all work in the direction of retention of water. That the body does not handle water normally in exacerbations of thyrotoxicosis, such as postoperative reactions, is indicated in the figures of Coller and Maddock (5) which show a gain in weight in the first and sometimes the second postoperative days at the time fever is increasing. We have long been impressed with the frequency in severe exacerbations, the spontaneous or postoperative "crisis in miniature," of profuse bronchial and tracheal mucous secretion amounting to "bronchorrhea," often with slight cyanosis, occurring in the absence of clinically demonstrable pulmonary consolidation in its early phases, with rapidly increasing fever and without x-ray evidence of atelectasis. Such patients do not have obvious heart

failure. We have seen prompt, though transient, benefit follow the administration of 6 per cent acacia solution and of transfusion of blood, both of which increase the circulating volume of blood by increasing the colloid osmotic pressure of the plasma and withdrawing water from the tissues. We have also found consultants reluctant to make a diagnosis of pulmonary edema at the onset of this usually fatal syndrome, but autopsy (which has not often been obtainable in patients dying of extreme thyrotoxicosis) has usually revealed gross pulmonary edema in them, in the experience of pathologists with whom the subject has been discussed, as well as in our own experience. Such a case has recently been commented on by Coller and Maddock (10). We are confident that we have initiated or hastened a fatal outcome in such patients by attempting to meet the usually accepted requirements for a large fluid intake, particularly in febrile reactions. The mechanism of fever production itself is not clear in this situation, any more than is its rapid onset in atelectasis and in surgical patients whose intra-abdominal and intrathoracic tension is suddenly increased by a too successful effort to close large ventral hernias without first reducing body weight. In all these cases, one is tempted to speculate that the *rapidity* with which vital capacity is diminished and presumably the total functioning area of alveolar epithelium is reduced, is perhaps through interference with the vaporization of water, related to the onset of fever.

We have also been concerned with the likelihood that there is edema of the brain in thyroid crisis. In 1932, the similarity between the clinical course of patients with severe cerebral injuries and those in thyroid crisis seemed so suggestive that we began to use 50 per cent glucose intravenously immediately after operation in poor risk patients (with a daily total fluid intake not usually exceeding 2500 c.cm.) in an effort to forestall severe postoperative reactions. We were very much gratified with the result and consistently observed less circulatory and febrile reaction than we would have, with confidence, counted on in patients so severely thyrotoxic. We followed that plan until the appearance of Frazier's advocacy of 10 per cent glucose for its effect in combatting the depleted glycogen reserves of the liver; Frazier used glucose in concentration of 10 per cent. We somewhat sheepishly concluded that our own patients had benefited merely from the liver-sparing effects of the glucose molecule. The era of large fluid intakes then succeeded, and the hopefulness of those—of whom the author was one—(2) who found further evidence of impaired liver function in thyrotoxicosis led also to efforts to get as much glucose as possible into the patient in the belief that the glycogen of the liver could thus be restored. Fluid intakes up to 5000 cubic centimeters daily, by all available routes, were freely advised, with, as we believe, often harmful results. More recent studies by a number of authors have indicated that there is no clear correlation between impaired liver function and the severity of the hyperthyroid reaction to operation and vice versa. In other words, impaired liver function is a "sideshow" and not the "main show." The occurrence of a phase of rising pulse pressure and fever, followed by a phase of falling pressure and vasomotor collapse, has long made us think that damage to nervous centers by whatever combination of edema, anoxemia, and acidosis is the essential and irreversible lethal process. Few authors today consider liver failure to be the cause of death in thyroid crisis. It is less widely recognized that large fluid intakes *per se* cause further depletion of liver glycogen (14) and that the mere administration of glucose does not actually cause restoration of liver glycogen.

These observations have renewed our interest in the question of edema of the brain. Autopsy material is scanty but Diamond has reported such pathological observations on 2 patients dying in thyroid crisis and Wolff records the experimental production of cerebral edema on the basis of lowered arteriovenous difference in oxygen content of the cerebral circulation. As noted, such a decreased difference in oxygenation of arterial and venous blood in the peripheral circulation has been demonstrated in thyrotoxicosis. For the past year we have resumed the prophylactic use of hypertonic solutions intraven-

ously and the limitation of total daily fluid intake to quantities between 2,000 and 3,000 cubic centimeters in severely hyperthyroid patients, particularly in the first 48 hours after operation. According to the criteria of Coller and Maddock this might be considered a dehydration regimen but we feel that the plan has merit. We have usually given 100 cubic centimeters of 50 per cent glucose intravenously twice daily on the day of operation and once on the first postoperative day. Hypodermoclysis of 1,000 to 1,500 cubic centimeters of mildly hypotonic glucose solution (2.5 to 5 per cent) is given following operation and the ingestion (or nasal catheter feeding) of such carbohydrate containing fluids as fruit juices and carbonated beverages, together with water, to an additional amount of 1,000 to 1,500 cubic centimeters of oral intake is planned for the day of operation. On the first postoperative day the oral intake usually suffices to make up the total intake. We make no effort to meet the total caloric requirement of these patients and have no objection to the combusion of their body fat during this period (with the precaution to be mentioned), since it is a much more concentrated source of energy than any with which we can provide them. In order to differentiate between the effect of hypertonicity of the concentrated glucose solution and the metabolic effects of the glucose molecule, *per se*, we have in the past year used 50 per cent sorbitol[1] intravenously in a few cases and find it quite as effective thus far in minimizing the severity of the postoperative reaction. It will be most interesting to observe the effect of transfusions of separated plasma in this situation, especially if concentrated preparations are used. Sufficient glucose must be given by other routes, however, to ensure that clinical acidosis does not occur, as unpublished studies (3) on the carbon dioxide combining power of the plasma and the excretion of total and organic acids of the urine strongly support the view that acidosis is an important feature of hyperthyroidism. These are in all probability ketone acids, since Somogyi has recently demonstrated severe cases of ketonemia in certain hyperthyroid patients.

[1] Sorbitol supplied through the kindness of the Abbott Laboratories.

SUMMARY

We believe that currently accepted figures for desirable fluid intake in surgical patients, especially for those with hyperthyroidism, will be revised downward.

All the mechanisms whose derangement in hyperthyroidism is partly understood seem to operate in a way to favor water retention by the tissues during exacerbations. Pulmonary and cerebral edema are apparently of frequent occurrence in crisis.

We have returned, with satisfaction, to a regimen of limitation of fluid intake and the use of hypertonic solutions intravenously in severely thyrotoxic patients.

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PRIMARY AND SECONDARY NEURILEMMOMA OF BONE

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ALTHOUGH the Registry of Bone Sarcoma has made provision in its newer classification for a wide variety of both benign and malignant tumors of bone and bone marrow, no provision has been made for the study of neurogenic tumors occurring in bone.

That nerve sheath tumors frequently originate in bone and periosteum is well attested by occasional communications appearing in the literature. Indeed, Geschickter devotes much space to a consideration of malignant neurogenic tumors and to the clinical and histological differentiation from other forms of fibrosarcoma.

There is a form of nerve sheath tumor whose specificity is assured by a distinctive histological appearance and clinical behavior which unfortunately is referred to by a bewildering variety of names indicating a diversity of opinion as to its histogenesis. Such a tumor has been described principally in the soft tissues of the body in direct continuity with peripheral or visceral nerves and has been termed peripheral neurinoma, peripheral neuroma, neurofibroma, perineural fibroblastoma, schwannoma and more latterly by Stout as a neurilemmoma.

In spite of an extensive survey of the literature dealing with this condition there has been only a single reference to the undoubted occurrence of this tumor in bone and that a brief case report by Gross, Bailey, and Jacox. These authors described a tumor involving the mid-shaft of the humerus in a 37 year old female with symptoms of 3 years' duration. The tumor was soft, solid, and the x-ray appearance had been cystic. Diagnosis was made after microscopic examination of the excised specimen, and the illustrations leave no doubt that this is a typical neurilemmoma. although the authors designated it as a neurofibroma.

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Peers described a cystic tumor of 1 year's duration located in the mid-shaft of the ulna in a 55 year old male and referred to it as a perineural fibroblastoma. Although the photomicrographs are not absolutely conclusive, they indicate, in conjunction with his microscopic descriptions, that his tumor would now be classified as a neurilemmoma. Peers described whorl-like fibroblastic areas with palisading of the nuclei but also described tumor giant cells and foam cells containing doubly refractile lipoid. Amputation was performed in the belief that the tumor was malignant. Twenty months following amputation, the patient was free of symptoms. The diagnosis of neurogenic sarcoma in Peers' case is open to serious question, as the author himself indicates.

It is most important in this connection to rectify an erroneous concept concerning the clinicopathological distinction of benign from malignant neurogenic tumors of bone. The concept is prevalent that once a neurogenic tumor invades bone, it is undoubtedly highly malignant and the possibility of cure is practically nil. Such a generalization is fallacious and will often lead to needless sacrifice of an extremity or to improper prognostication. Both of the tumors herein reported showed extensive bone destruction yet were undoubtedly benign. This is not intended to imply that malignant neurogenic sarcomas of bone do not occur. Every case must be decided on its own merits. Mere bone invasion by a neurogenic tumor is *per se* inadequate to insure the diagnosis of malignancy.

Perhaps the most comprehensive of the scattered references to the specific nerve sheath tumor is that of Stout who reported 52 tumors in peripheral locations and collected 194 authentic cases from the literature. Stout gives Verocay credit for having been the first author who adequately described the tumor and distinguished it from other neurogenic tumors. In Stout's combined series of 246 cases there was no single instance of the

occurrence of the tumor in bone. He further found stigmas of von Recklinghausen's disease in 18 per cent of 50 cases and believed that the co-existence of neurilemmoma and von Recklinghausen's disease might have been higher if the stigmas had been more carefully noted. Frequently a few pigmented cutaneous areas are the only outward manifestation of von Recklinghausen's disease. In his analysis, many of the tumors involved the extremities and almost all of these occurred on the flexor aspects of the extremities where the larger nerve trunks were present. Their localization was concentrated near the elbow, wrist, and knee. The ankle and hand were also occasionally involved. Clinically, the diagnosis was rarely made before operation, the tumor usually being classified a cyst, fibroma or, if it involved the hand, a ganglion. Stout found a number of such tumors involving the neck, face, scalp, trunk, eye and orbit, the upper respiratory tract and 35 cases involving the stomach. Within the thorax, neurilemmomas have been reported as occurring in the posterior mediastinum.

PATHOLOGY

More complete descriptions are to be found in general treatises. The tumor grossly is encapsulated, the capsule being formed by the epineural sheath and is attached to a nerve which may be so small as to be overlooked by the surgeon. The shape is rounded or fusiform. The largest tumor in Stout's series was 6 centimeters in diameter. The tumors are always slow growing, frequently soft and fluctuant, and have a pink to gray appearance. Often they are extremely vascular.

Stout, following Antoni, distinguished two types of tissue called for sake of simplicity, types A and B. Type A tissue is extremely specific in its appearance. The nuclei have a characteristic orientation about thin wire-like fibrils which is described as palisade formation. Type B tissue consists of a loose reticular arrangement of cells and fibrils in haphazard formation. The fibrils are often widely separated about microcystic spaces. The type B tissue is often extremely vascular and sometimes presents an angiomatoid appearance resembling fibro-angioma. Transitions from A to B types exist throughout the tumor. An isolated unit of type A tissue is often referred to as a Verocay body. The presence of occasional foam cells such as are observed in fibroblastoma of the spinal cord and nerve sheath does not exclude the diagnosis of neurilemmoma although Stout found them only exceptionally in his tumors.

SYMPTOMS

In Gross' case, the symptoms were merely the presence of a painless mass over the middle of the left humerus of 3 years' duration. X-ray disclosed a cystic lesion involving the medulla and cortex. There was no evidence of generalized neurofibromatosis. In our first case 5 pathological fractures of the ulna had occurred over a period of 8 years before the patient consented to a resection of the tumor. Pain was not a symptom, however. In our second case painful pressure symptoms preceded the discovery of the tumor by 2½ years. The tumor originated from the fifth lumbar nerve within the neural canal and filled a wide excavation of the upper sacrum. Origin of the tumor in the first case was probably from an intracortical nerve. In this connection, it would seem advisable to review the existing concepts concerning the nerve supply to bone.

THE NERVE SUPPLY TO BONE

The absence of sizable nerve trunks leading into bone together with the well known relative analgesia of osseous tissue, has created a prevalent concept that bone is without nerve supply. This concept is erroneous. Nerve tissue has been repeatedly observed to be present more or less constantly in both the compact and cancellous bone of man.

Among the earlier histologists, Luschka, Kobelt, Engel, Beck, and Kolliker, all observed and recorded the presence of both a cerebrospinal and a sympathetic nerve supply to the bones of the extremities, vertebræ and skull. Kolliker, writing in 1852, gave a detailed description of the nerves of the osseous system, which he observed as being derived both from the richly innervated periosteum and as one or more minute trunks accompanying the nutrient vessels.

More recently, Stohr, writing in Mollendorff's comprehensive handbook, noted the presence of these 2 sources of nerve supply to bone, and illustrated a fine nerve trunk passing through bony tissue. According to Stohr, the periosteum is abundantly supplied with nerves. From these, minute fibers enter through Volkmann's canals into the underlying bone. Ramifications and anastomoses are scanty and no definite nerve endings have been consistently identified. It has been suggested that they are gradually attenuated and may terminate as free extremities. Maximow has described these "free endings" in connective tissue wherein the nerve fiber first loses its myelin and then, being accompanied only by Schwann's nuclei, breaks up into thin, terminal threads.

The nerves accompanying the larger blood vessels to the bones are small and usually can be identified as a ramifying plexus about these vessels. Definite independent nerve trunks, while uncommon, have been observed. These nerves are largely of autonomic origin. No definite nerve endings from this source are observed in the medullary tissues. Any of the neoplasms derived from peripheral nerve fibers therefore may conceivably arise from these nerves in bone.

CASE HISTORIES

CASE 1. K. S., male, aged 37 years. Diagnosis: primary neurilemmoma arising in midshaft, right ulna, duration 8 years.

In November, 1931 following a mild trauma the patient sustained a pathological fracture through the shaft of the right ulna. Prior to this time the arm had been symptom-free. X-ray examination revealed the fracture through a circumscribed area of bone destruction which was considered to be a solitary bone cyst. Healing in a cast was uneventful.

During the course of the next 7 years the right ulna was refractured four times through the same site. Upon reduction and cast immobilization, the fractures, had, in each instance, healed in approximately normal time. Following the initial fracture, a firm, fixed, non-tender mass was noted on the ulnar shaft at the fracture site. The mass had gradually increased in size with each additional injury. Except for moderate weakness and ache on prolonged use, the forearm was symptom-free between episodes of fracture.

The patient was first seen at the Hospital for Ruptured and Crippled September 17, 1939, after having fractured the ulna for the fifth time. A firm mass of bony hardness was present over the midshaft of the right ulna. Tenderness to pressure was noted, and slight false motion was present, causing pain. Wrist and elbow motions were normal except for 10 degrees of limitation of forearm supination. Moderate soft tissue atrophy of the extremity was present. Regional lymph nodes were normal. Examination of the osseous system elsewhere was negative. General examination revealed no stigmas of von Recklinghausen's disease, and routine laboratory examinations were not contributory. One sister died of a brain tumor of undetermined type. The family history was otherwise negative.

X-ray examination showed a pathological fracture through an area of multilocular cystic change in the middle of the ulnar shaft. The cystic area had expanded the anterior cortex of the ulna to a marked degree, and had completely eroded the lateral cortex, leaving an incomplete, thin cystic shell of cortical bone.

On September 18, 1939, the forearm was explored under tourniquet hemostasis. Upon exposure of the ulna, a firm, fusiform enlargement was encountered, measuring 2 by 4 centimeters. The periosteum was intact over the anterior, medial, and posterior surfaces. On the lateral surface the bone and overlying periosteum were perforated by tumor tissue which extended into surrounding muscle. No nerves could be identified passing into the tumor mass.

The entire tumor was dissected free from the surrounding tissues and resected together with 1 centimeter of ulnar shaft on either side. The 6 centimeters defect in the ulna was bridged with a massive tibial bone graft. The wound was closed without drainage and a cast was applied. Convalescence was uneventful and there has been no evidence of recurrence to date.

Gross pathological examination of the tumor showed it to be soft, hemorrhagic, and of irregular consistence. Some areas were almost gelatinous while others were gritty. The tumor appeared to arise from and to grow out of the shaft of the ulna. Microscopic examination revealed a typical neurilemmoma, and the diagnosis was confirmed by Stout, Foot, and Symmers.

CASE 2. S. Mac I., female, aged 42 years. Diagnosis: secondary neurilemmoma arising from dorsal root of fifth lumbar nerve, right, with invasion of right sacral wing; duration, 3 years.

Symptoms started in the summer of 1936 with acute onset of pain in the right leg and buttock. There was no history of preceding injury. The pain first appeared after patient had stood several hours on a hard, uneven surface. A cramp-like pain in the calf muscles was most severe at the onset and gradually extended throughout the entire posterior leg from buttock to foot. The pain was continuous, and required sedatives at night.

On October 18, 1939, the patient first attended the low back clinic at the Hospital for Ruptured and Crippled. She had received treatment almost continuously from the time of onset of pain without

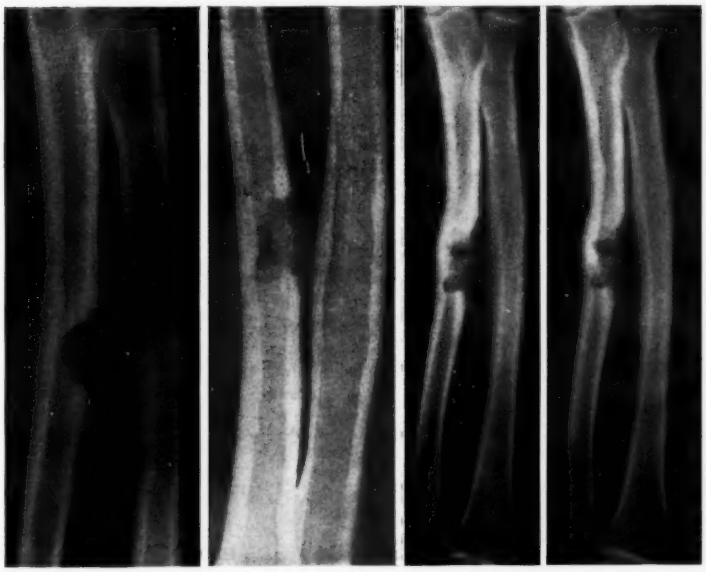

Fig. 1. Fig. 2. Fig. 3 a and b.

Fig. 1. Neurilemmoma mid-shaft of right ulna, Case 1, July 18, 1932.
Fig. 2. Roentgenogram of Case 1, taken May 21, 1935.
Fig. 3. a and b, Roentgenogram of Case 1 taken September 17, 1939, following a spontaneous pathological fracture through the site of disease.

relief. Injections, physiotherapy, corset supports, osteopathy, and a series of 10 artificial fever treatments had been of no avail. Roentgenograms of the low back made at another hospital in 1937 were considered negative.

Examination showed a small woman, standing erect, with good posture and without a list. Leg lengths were equal. Straight leg raising was limited on the right. Forward bending was moderately limited. Sacro-iliac tests were negative. Right Ober sign was positive. There was tenderness to pressure in the right iliolumbar angle which extended to the right sacrosciatic notch.

Neurological examination revealed an absent right Achilles reflex. All other tendon reflexes were normal. Sensation to pin and light touch was diminished over the dorsal aspect of the right calf and foot. No muscular atrophy was present. General examination was negative.

Laboratory findings were non-contributory with the exception of a serum alkaline phosphatase of 15.0 Bodansky units. Spinal fluid examination was negative.

The patient had undergone a thyroidectomy for toxic goiter several years prior to her present illness. In addition, several small tumors had been recently removed from the subcutaneous tissue of the right leg. These had been diagnosed[1] as benign fibro-

[1]By Dr. N. C. Foot.

Fig. 4. Drawing of gross specimen in Case 1 which shows the resected portion of the shaft of the ulna with the tumor occupying the medullary canal and the lateral subperiosteal area. The pathological fracture can be seen in the illustration.

Fig. 5. Photomicrograph through an area of the tumor, Case 1. Low power showing type A tissue with characteristic nuclear palisading.

Fig. 6. Photomicrograph through another area of tumor, Case 1. Low power showing type B tissue.

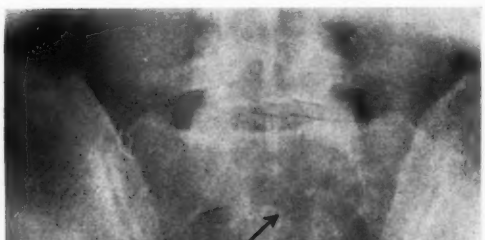

Fig. 7. Roentgenogram of the sacrum in Case 2 showing an irregular destructive bony defect occupying a portion of the middle and right upper sacrum.

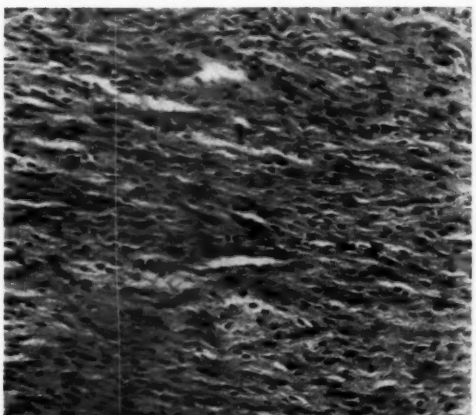

Fig. 8. Photomicrograph of a section of the tumor removed from Case 2. Low power, type B tissue.

Fig. 9. Same case, low power, characteristic type A tissue.

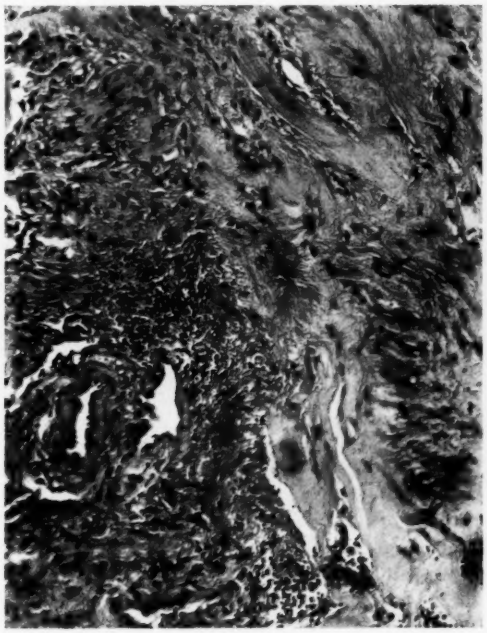

Fig. 10. Same case, low power, ligamentum flavum on right invaded by type B tissue on left.

angiomas and re-examination confirmed that diagnosis. These tumors were seemingly unrelated to the present disease.

X-ray examination showed a large irregular bony defect present in the right side of the upper two sacral segments. Accordingly, admission was advised.

Two aspiration biopsies failed to establish the diagnosis and on November 24, 1939, an open biopsy was done. On section the tissue was a typical neurilemmoma.

On December 8, 1939, excision of the tumor was performed. A right hemilaminectomy of the fourth and fifth lumbar vertebræ and upper right sacrum was done, exposing the tumor, which was a multilocular, largely circumscribed mass about 4 by 6 centimeters, although in one area it had invaded the ligamentum flavum. A large, irregular, smooth walled cavity of corresponding size was present in the right sacral wing extending laterally to the right sacral crest and medially across the midline. The cavity was filled with hemorrhagic, gelatinous tumor tissue through which the upper three right sacral nerves passed. The tumor was removed piecemeal, it being necessary to sacrifice the second sacral nerve-root.

The patient has experienced an uneventful convalescence and 2 months following removal has been able to resume her normal activities. Occasional momentary lightning-pains down the right leg in association with mild hyperesthesia over the posterior thigh and calf comprise her only symptoms.

Microscopically, the tumor was a typical neurilemmoma which was locally invasive, confirming that observation at operation.

Our Case 1 is strikingly similar to Gross' and Peers' cases in anatomical and x-ray appearance. Both involved the mid-shaft region of the bone replacing portions of cortex and medullary cavity. Gross' tumor was removed by local excision, whereas our tumor was treated by resection of a portion of the ulna. Finger-like processes of tumor tissue extended into the cortical bone in such a fashion as to leave doubt that simple excision would have proved adequate.

Our Case 2 was non-encapsulated and invaded the bony sacrum indicating that occasionally the tumor may be locally invasive. Indeed, it even infiltrated the ligamentum flavum. Because of its location, complete removal was not possible; hence recurrence is anticipated.

X-ray examination in both cases revealed cystic lesions with punched out cortical rarefactions in the first case.

RELATIONSHIP OF NEURILEMMOMA OF BONE TO NEUROFIBROMA OF BONE AND VON RECKLINGHAUSEN'S DISEASE

In neither of the 2 cases nor in those of Gross and Peers were stigmas of von Recklinghausen's generalized neurofibromatosis found.

It is well known as a result of the investigations of Adrian, Stahnke, Brooks and Lehman, and Nørgaard to mention but a few, that osseous changes may play a paramount rôle in von Recklinghausen's disease. These changes include a variety of lesions and will be mentioned only briefly here to avoid confusion with the neoplastic lesion under consideration. Scolioses, lordoses, unilateral hypertrophy of an extremity, localized cortical and subperiosteal rarefactions, unexplained osteoporoses with pathological fracture and resultant pseudoarthroses occur so frequently as often to overshadow the cutaneous manifestations of the disease. In general, these lesions are all of two varieties: (1) skeletal changes due to faulty embryogenesis and (2) skeletal changes due to osseous neurofibromatosis. Biopsies of the periosteal or cortical cystic lesions have been performed in a few instances, notably in one of the cases of Brooks and Lehman in which a cystic lesion in the left tibia was found to be a neurofibroma associated with systemic manifestations of von Recklinghausen's disease. Similar roentgenological lesions have been seen by a number of observers leading to the conclusion that the frequency of their discovery is closely related to the thoroughness with which the body skeleton is roentgenographed.

The roentgenological manifestations of neurilemmoma of bone are strikingly similar in the 3 cases. In Gross' case, a cystic lesion of the mid-humerus, in Peers' and our Case 1, a cystic lesion of the mid-ulna. The localization in the mid-shaft region may be a coincidence or may be related to the anatomical distribution of the nerves in bone. Presumably the more sizable nerve trunks enter the periosteum in the diaphyseal region along with the nutrient vessels.

Differential diagnosis from neurogenic sarcoma is established by the sharp, clear character of the x-ray defect, the mildness of pain and the long duration of symptoms.

From osteitis fibrosa cystica, the differential diagnosis is made principally by the age of the patient, the cysts of the latter occurring generally before the twentieth year. From chondroma differential diagnosis is not easy. These defects, however, are more prone to involve the ends of the bones and are located principally in the phalanges, ribs, and sternum. Stigmas of osteochondrodystrophy are apt to be present in chondroma although not necessarily so.

SUMMARY AND CONCLUSIONS

1. Neurilemmoma is a specific form of nerve sheath tumor which may occasionally occur as a primary tumor of bone. The origin of the tumor in one of our cases and in the 2 cases in the literature is probably from nerve sheaths in the cortex, although they may arise primarily in the periosteum and secondarily invade the bone.

2. Neurilemmoma may invade and excavate bone by direct extension from a paraosseous focus as in Case 2 in which a large tumor of the fifth lumbar nerve produced an extensive defect in the sacrum.

3. Bone invasion by a neurogenic tumor, therefore, does not *per se* indicate a sarcomatous transformation of a benign tumor, and such a concept is fallacious and fraught with considerable danger.

4. Neurilemmoma of bone is not to be confused with the less specific neurofibromas that have been reported in bone in association with systemic and skeletal manifestations of von Recklinghausen's disease.

5. The proper treatment of neurilemmoma of bone would appear to be conservative. Thorough curettage with cautery or subperiosteal resection and bone grafting would appear to be adequate.

6. In consideration of cystic expansive rarefactions in the mid-shaft region of long bones, the possibility of neurilemmoma should be considered.

The authors wish to express their thanks to Drs. T. Nicola and E. E. Meyers for permission to report the 2

cases and to Dr. A. Purdy Stout for his helpful suggestions and criticisms.

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A COMPARISON OF THE IRVING AND POMEROY METHODS OF TUBAL STERILIZATION

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SINCE 1850 many different operations for sterilization have been performed on the fallopian tubes in an attempt to find a technique that would prove to be 100 per cent successful. First, simple ligation was tried, then double ligation with section of the tube between the ligatures. Various methods have been reported of burying the distal portion of the cut tube either in the mesosalpinx or uterine musculature. The most popular techniques in the past 20 years have been the Madlener, the Pomeroy, also known as Pomeroy-Bishop, or Pomeroy-Lull, and the wedge-shaped excision of the tubes, including the cornua.

The Pomeroy technique consists of picking up a loop of the middle portion of the tube, ligating it with an absorbable suture and then resecting the loop. Lull reported 223 cases of sterilization by this method with no known subsequent pregnancies, although, he had a follow-up of only 69 per cent of his cases. Bishop (2) and Bishop and Nelms (1) report from the Brooklyn Hospital 319 cases of successful sterilization by the Pomeroy method. Lutz reported one case of ectopic pregnancy in the distal portion of the tube following a Pomeroy sterilization.

There is one patient (G. O.) who entered the Boston Lying-in Hospital for delivery who gave a history of having had a sterilization operation performed the year before in a small hospital in Texas. A cesarean section was performed and a bilateral excision of the tubes with a wedge-shaped resection of the cornua was done at our Hospital. Both tubes showed a thinned-out portion approximately 1.5 inches from the cornua and about 1.5 inches long; the site of previous ligation. No account of the operation could be obtained from the hospital, or the doctor in Texas, but it was thought to be a Pomeroy operation or a partial tubal resection with subsequent pregnancy.

Dr. F. S. Kellogg tells me of a patient of his (H. J.) with a Pomeroy sterilization in 1932, followed by a pregnancy occurring 1 year later. There is one case (A. H.) in the Boston Lying-in Hospital records of failure to sterilize by this operation. This patient, who had rheumatic heart disease, had a cesarean section and Pomeroy sterilization in 1933. The following year she returned 4 months' pregnant, and had an abdominal hysterotomy and resection of the right tube well into the cornu of the uterus and excision of the proximal portion of the left tube, including the uterine cornu. The right tube was studied microscopically and found to be normal with the exception of an area in the thinned out fibrous portion of the tube just proximal to the point

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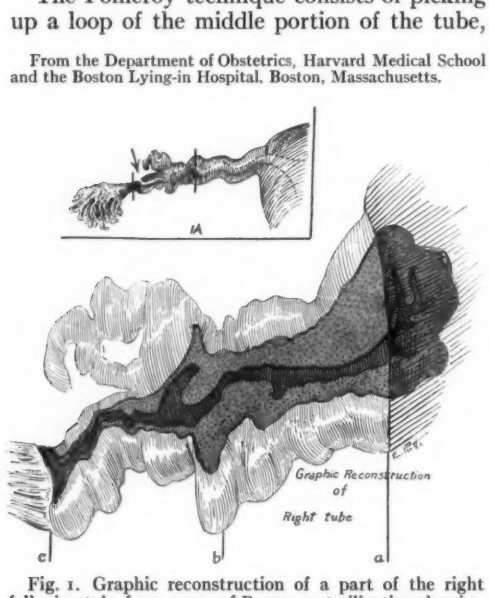

Fig. 1. Graphic reconstruction of a part of the right fallopian tube from a case of Pomeroy sterilization showing the connection by sinus formation between the lumen and the peritoneal cavity. ×60. a, The level of section shown in photomicrograph Figure 2; b, represents Figure 3; c, represents Figure 4. The arrow shown in Figure 1A represents the point of ligation of the tube at the time of operation. The block of tissue studied was just proximal to this point.

Fig. 2. Cross section of the right fallopian tube from a case of Pomeroy sterilization. The position of this section is represented in Figure 1 by line *a*. Tubal lumen almost normal in size and shape. Hematoxylin and eosin stain. ×75.

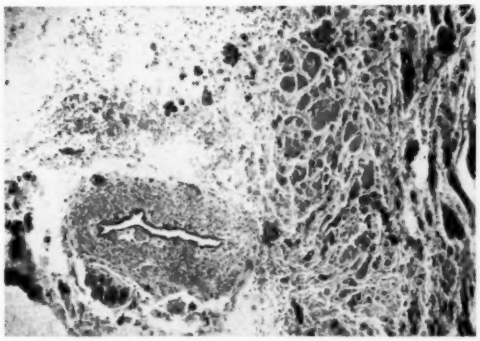

Fig. 3. Cross section of the right fallopian tube from a case of Pomeroy sterilization. The position of this section is represented in Figure 1 by line *b*. Tubal lumen greatly reduced and tubal wall very thin and lying near peritoneal surface of tube. Hematoxylin and eosin stain. ×75.

of previous ligation. Serial microscopic sections of this tissue were studied and a graphic reconstruction made (Fig. 1).[1] Photomicrographs of representative sections are shown in Figures 2, 3, and 4. Here can be clearly demonstrated a small epithelium-lined sinus open to the peritoneal cavity and continuous with the tubal lumen. Through this sinus the ovum might have gained entrance into the fallopian tube. Microscopic study of the left tube shows a similar sinus connecting the peritoneal cavity and the lumen of the fallopian tube. Photomicrographs of representative sections of this block of tissue are shown in Figures 5, 6, and 7.

The Madlener technique consists of lifting up a loop of the fallopian tube at about its middle point and crushing it with a clamp. The clamp is removed and replaced by a ligature of non-absorbable material. Madlener, in 1919, reported 89 cases of successful sterilization by this method. Von Graff, in bibliograph in 1939, reports 304 cases of sterilization by the Madlener technique with only one subsequent pregnancy. He has made a study of the literature since 1919 and finds 3,975 cases reported of sterilization by the Madlener technique with only 18 failures. Thus with his own cases he reports a total of 4,279 cases with 19, or 0.44 per cent failures.

In the 21 years from 1916 to 1937, there have been 645 operations for sterilization performed in the Boston Lying-in Hospital. These were done by the following methods: Irving, 432; hysterectomy, 93; Pomeroy, 50; wedge-shaped excision of tubes at the cornua, 50; oophorectomy, 6; salpingectomy, 5; defundation of the uterus, 2; ligation of tubes, 2; and unilateral Irving combined with one of the other above methods, 5. It is interesting that of these patients 388, or 60.2 per cent,

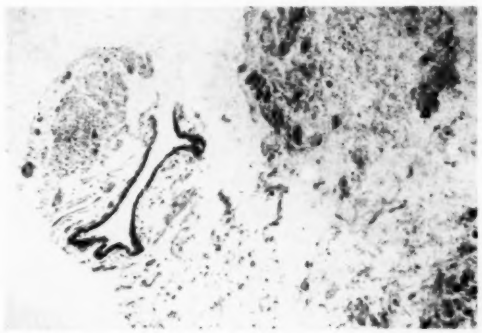

Fig. 4. Cross section of the right fallopian tube from a case of Pomeroy sterilization. The position of this section is represented in Figure 1 by line *c*. Tubal lumen open to the peritoneal cavity. Tubal wall consists mostly of mucosa with a small amount of connective tissue surrounding it. Hematoxylin and eosin stain. ×75.

[1]Method of graphic reconstruction: Serial microscopic sections were studied and camera lucida tracings made on thin translucent manifold bond paper. Orientation of these tracings was done by piling them one on top of the other on a table possessing a ground-glass top illuminated from below. Permanent orienting lines intersecting at right angles were then successively drawn on each tracing starting with the lowest. A theoretical plane of section at a right angle to the actual plane of section was chosen. The structures of the individual sections lying in this theoretical plane were plotted on graph paper, the horizontal orienting line being used as a base line.

Fig. 5. Cross section of left fallopian tube from a case of Pomeroy sterilization. This is from same case used in Figures 1, 2, 3, and 4 but the other tube. This section is through the whole tube and surrounding scar tissue. Closed tubal lumen and thin tubal wall are seen in the lower right corner. Hematoxylin and eosin stain. ×18.

Fig. 6. Cross section of the left fallopian tube from a case of Pomeroy sterilization. This is from the same case used in Figures 1, 2, 3, and 4 but the other tube. The thin walled tubal lumen with a small sinus opening from it into the peritoneal cavity is seen in the lower right corner. Hematoxylin and eosin stain. ×18.

were Roman Catholic; 178, or 27.6 per cent, were Protestant; 68, or 10.5 per cent, were Jewish; and 11, or 1.7 per cent, were Greek Orthodox.

The medical indications for sterilization were as follows: rheumatic heart disease, 208; previous cesarean sections, 152; chronic nephritis, 58; pulmonary tuberculosis, 42; diseases contra-indicating future pregnancies, 42; toxemia, 23; hypertension, 23; relaxed pelvic floor, 22; deformities or diseases of the pelvic soft tissues, 23; abnormalities of pregnancy or labor, 18; deformities or diseases of the bones, 17; and mental deficiency, 17.

Fig. 7. Cross section of the left fallopian tube from a case of Pomeroy sterilization. This is from the same case used in Figures 1, 2, 3, and 4 but the other tube. The tubal lumen open by a wide sinus into the peritoneal cavity is seen in the lower right corner. Hematoxylin and eosin stain. ×18.

By the Irving method of sterilization one horn of the uterus is drawn into the abdominal wound. The tube is picked up by a bite of a clamp and traced down to its fimbriated extremity to prevent any error in identification between it and the round ligament. One and one-half inches from the cornual insertion a ligature of No. 1 chromic catgut is passed through the mesosalpinx and tied. Just proximal to this a double suture ligature of the same material, mounted on a half curved, round pointed needle, knotted about 3 inches from the free ends, is tied about the tube. This suture ligates the tube proximally and serves as a tractor in the steps which are to follow (Fig. 8). The mesosalpinx is clamped with two fine pointed hemostats and the tube is cut free. The mesosalpinx is tied with a No. 0 chromic catgut ligature (Fig. 9). A stab wound is made into the muscularis at the base of the proximal end of the tube. A sharp pointed hemostat is plunged into the wall of

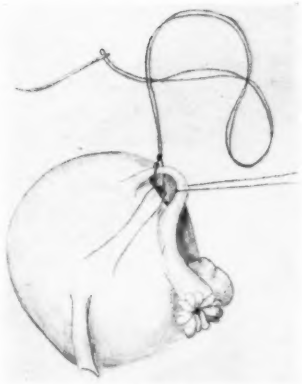

Fig. 8.

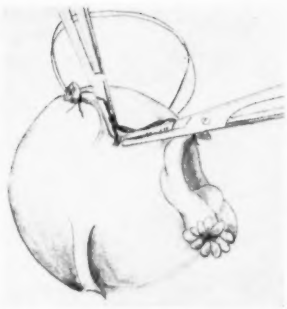

Fig. 9.

Fig. 10.

Fig. 8. First stage of sterilization operation by the Irving method. A ligature of No. 1 chromic catgut is passed through the mesosalpinx and tied. Proximal to this a double suture ligature is tied about the tube.

Fig. 9. Second stage. The mesosalpinx is clamped with two fine pointed hemostats and then the tube is cut free.

Fig. 10. Third stage. Irving method. A stab wound is made into the muscularis and a hemostat is plunged into the wall of the uterus through this wound and the point is spread, thus making a pit in the uterine wall.

the uterus through this stab wound as far as its lock and the point spread thus making a pit in the uterine wall (Fig. 10). A grooved director is passed into this pit as far as it will go and the half-curved needle on a needle holder is passed along the director into the extreme depth of the pit and brought out on the surface of the uterus (Fig. 11). The director is then removed and traction on the suture ligature causes the tube to enter the pit in the uterine wall for a distance of approximately three-quarters of an inch (Fig. 12). One strand of the double suture ligature is cut and a cross stitch is made in the superficial portion of the uterine wall and the free ends are tied together, thus anchoring the tube in the depths of the pit. The small wound at the point of entry of the tube is closed with a figure eight suture of No. 1 chromic catgut, thus completing the operation (Fig. 13). The

Fig. 11.

Fig. 12.

Fig. 13.

Fig. 11. Fourth stage of sterilization operation by the Irving method. A grooved director is passed into the pit as far as it will go and a half curved needle is passed along the director and brought out on the surface of the uterus.

Fig. 12. Fifth stage of sterilization operation by the Irving method. Traction on the suture ligature causes the tube to enter the pit in the uterine wall for a distance of approximately three-quarters of an inch.

Fig. 13. Sixth stage of sterilization operation by the Irving method. The tube is anchored in the uterine wall.

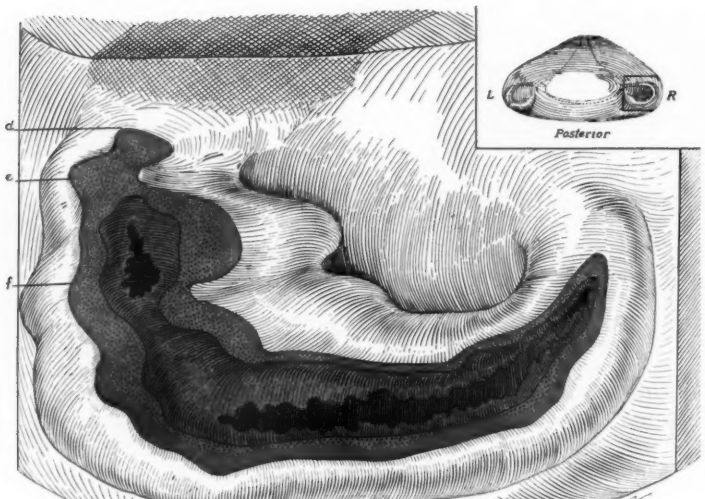

Fig. 14. Graphic reconstruction of right cornu of uterus from a case of Irving sterilization viewed from above, the anterior surface of the uterus lying to the right. The reconstruction was made at such a plane as to include the lumen. The cross-hatched area at the upper left represents the scar tissue in the uterine wall on the cut surface of the block of tissue at the point where the tube is buried in the uterine wall. *d*, Represents the position of the section seen in photomicrograph Figure 15; *e*, represents Figure 16; *f*, represents Figure 17. Inset shows the position of the reconstructed block in relation to the uterus.

distal end of the tube is left free in the peritoneal cavity. The same procedure is repeated on the other side.

We have studied the uterus and tubes from a patient (G. J.) who, in 1928, had a sterilization operation performed by the Irving tech-

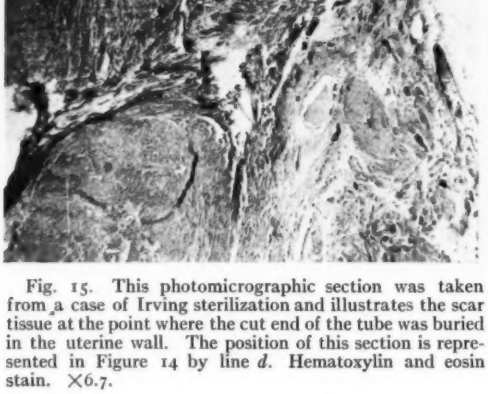

Fig. 15. This photomicrographic section was taken from a case of Irving sterilization and illustrates the scar tissue at the point where the cut end of the tube was buried in the uterine wall. The position of this section is represented in Figure 14 by line *d*. Hematoxylin and eosin stain. ×6.7.

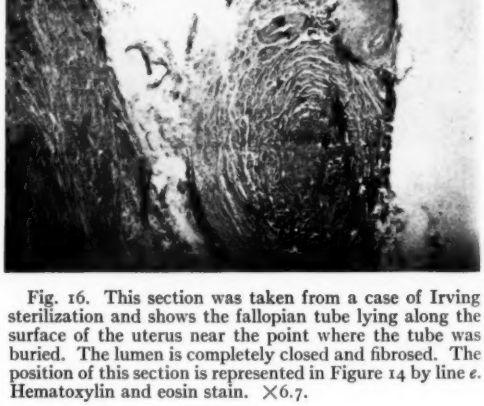

Fig. 16. This section was taken from a case of Irving sterilization and shows the fallopian tube lying along the surface of the uterus near the point where the tube was buried. The lumen is completely closed and fibrosed. The position of this section is represented in Figure 14 by line *e*. Hematoxylin and eosin stain. ×6.7.

nique. Eight years later a supracervical hysterectomy and bilateral salpingo-oophorectomy were done because of fibroid tumors. The right tube was sectioned and these serial sections have been studied and a graphic reconstruction made (Fig. 14). This shows the way the tube curves and is finally buried in the uterine muscle; its lumen is patent except for that portion adjacent to the uterine muscle where it merges with the scar tissue formed at the site of incision. Photomicrographs of three representative sections are shown in Figures 15, 16, and 17.

Of the 432 cases in which sterilization was done by the Irving technique, we have found no instances of subsequent pregnancy. We have not attempted to communicate by letter or interview all these patients, but feel confident that in case of a pregnancy following sterilization, the patient would communicate with us. There was one patient (H. K.) who, because of rheumatic heart disease, was sterilized by the Irving technique in December, 1927. She was re-admitted in November of the following year because she thought she was pregnant. She gave a history of missing her June menstrual period, then having normal periods in July and August. Her September period lasted 7 days and was followed by a free interval of 12 days after which she began to have a scanty, brownish discharge which continued until 2 days before admission. She had noted some enlargement of her abdomen. Physical examination confirmed the diagnosis of rheumatic heart disease. By rectal examination a mass was felt on both sides of the uterus and the diagnosis of uterine fibroids was made. She refused operation and was discharged. No Aschheim-Zondek test was made.

There is a case of failure to sterilize by operation on the fallopian tubes which is of interest because the technique rather than the method was at fault. This patient, (H. K.) because of a small pelvis, was delivered by cesarean section in 1928. In 1933, she had a second cesarean section and at this time was sterilized, by means of the Irving method on the left side, and the Pomeroy method on the right side where dense, fibrous adhesions were found. She returned in 1934, pregnant, at term, and was again delivered by cesarean section. Her right tube appeared normal and there was a corpus luteum in the right ovary. The entire right tube, the distal portion of the left tube, and a block of the uterus including the left cornu and the buried end of the left tube were excised. On pathological examination the proximal portion of the left tube, the site of the Irving sterilization, was found to be buried in the uterine musculature and the lumen was closed. The right tube was found to be entirely normal. The microscopic sections made from the tissue submitted as right tube at the time of her first attempted sterilization were then re-examined. This tissue was seen to be composed of fibrous tissue containing bundles of myometrium, which at the time of the first examination had been thought to represent the muscular wall of the tube. It is probable that at her first operation a dense, fibrous adhesion was mistaken for the tube and ligated and cut.

Fig. 17. This section was taken from a case of Irving sterilization and shows the normal fallopian tube lying along the surface of the uterus. This section is represented in Figure 14 by line *f*. Hematoxylin and eosin stain. ×6.7.

SUMMARY

The results of sterilization by the Pomeroy and the Madlener operations on the fallopian tubes have been discussed.

The Irving technique of sterilization by operation on the fallopian tubes has been described.

One case of failure to sterilize by the Pomeroy method has been shown by microscopic study to be due to re-establishment of

the tubal lumen bilaterally by the formation of sinuses opening into the peritoneal cavity.

One patient sterilized by the Irving method 12 years previously has been shown to have the tubal lumen occluded. Serial microscopic sections of this tube were studied and a graphic reconstruction made.

Of 432 operations on the fallopian tubes for the purpose of sterilization there were no failures by the Irving technique; of 50 cases there was one failure by the Pomeroy technique.

I wish to express my appreciation to Dr. Frederick C. Irving for the use of his hospital records and statistics and for his work in describing and illustrating his operation on the fallopian tubes. I also wish to express my appreciation to Dr. Arthur T. Hertig for his kindness and assistance in making the photomicrographs used in this paper, also for teaching me the method of graphic reconstruction, and to the technicians of the pathological department of the Boston Lying-in Hospital for their assistance.

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THE NERVOUS FACTOR IN TRAUMATIC SHOCK

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In recent years traumatic shock has come to be regarded as largely, if not entirely, due to fluid loss into the tissues at the site of trauma. We are indebted chiefly to Blalock (2), and Parsons and Phemister for an awareness of the importance of this factor. Since the appearance of their work in 1930, other possible factors have been given little consideration. It was not until very recently that Slome and O'Shaughnessy published the results of what were probably the first experiments in which the rôle of the nervous system in the etiology of traumatic shock was demonstrated under carefully controlled conditions.

These investigators, after isolating the hind limb of a cat from the general circulation, leaving the principal nerves intact, perfused it from the circulation of another animal, or by means of a pump. Trauma to such a limb resulted in shock, while in control animals in which the nerves were severed, shock did not develop. Trauma consisted of twenty blows with a heavy iron rod, breaking the femur and damaging the muscles of the thigh. Transfusion in the shocked animals was found to have only a transient effect, whereas if accompanied by spinal anesthesia, benefit was more lasting. In a previous paper, O'Shaughnessy and Slome had reported that spinal anesthesia significantly delayed the onset of traumatic shock in the intact cat under chloralose anesthesia. Recently some of the work of O'Shaughnessy and Slome has been called into question by other investigators. Bell, Clark, and Cuthbertson repeated the cross-circulation experiments as described by Slome and O'Shaughnessy, and found that, although shock did occur in the experimental animals, all of the donor cats died first. They did note, however, in a number of cats under chloralose anesthesia, that shock was delayed in animals in which the traumatized limb was separated from the body except for the femoral vessels, as compared with animals in which the major nerves to the limb were left intact. Blalock and Cressman (3) also reported unsatisfactory results from cross-circulation experiments. However, in the intact cat under chloralose anesthesia, some increase in the period of survival following trauma was observed when spinal anesthesia was also induced. These results could not be reproduced when nembutal was substituted for chloralose. These investigators reported abrupt, terminal drops in blood pressure and deaths apparently due to respiratory failure when chloralose was used.

The studies to be described were undertaken in an effort to resolve some of the contradictions surrounding this phase of the problem of experimental traumatic shock.

METHODS

The studies were carried out entirely on cats. The anesthetics employed were nembutal in doses of 25 milligrams per kilogram, chloralose, 80 milligrams per kilogram, intrathecal procaine, 1.7 milligrams in 0.05 cubic centimeter of water per kilogram, and ether. Tracheotomy was done routinely and scrupulous care, particularly in those animals in which ether was used, was taken to keep the air passages free. In most experiments blood pressure was recorded continuously from a cannula in a carotid artery by means of a mercury manometer. Chlorazol fast pink (5 per cent), injected into the tubing just above the cannula in 0.5 cubic centimeter quantities every half hour, served as the anti-coagulant (8). This method was resorted to after it had become clear that the use of citrate was materially altering the course of the experiments. Body heat was maintained in every case by either an electric heating pad or a heat cradle. Rectal temperature was measured continuously. At the close of each experiment the animals were carefully killed for autopsy,

From The Department of Physiology, University of Minnesota.
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TABLE I.—TRAUMATIC SHOCK IN THE INTACT CAT

No. Sex	Weight kgm.	Anesthesia	Procedure	Course	*Adventitious pathology	†Fluid loss in per cent body weight
1 F.	2.5	Nembutal 28 mgm. per kgm.	20 blows to thigh. Carotid pressure. Citrate anticoagulant	Blood pressure to 200 following trauma, followed by a sharp fall to 0 in a few minutes. The fall was broken by several large swings upward	Multiple fractures, femur	0.56
2 F.	2.0	Nembutal 25 mgm. per kgm.	20 blows to thigh. Carotid pressure. Citrate anticoagulant	Blood pressure decline beginning 2½ hours after trauma, and reaching 0 in 1½ hours. Survival, 4 hours	Multiple fractures, femur	1.9
4 M.	2.0	Nembutal 25 mgm. per kgm.	20 blows to thigh. Carotid pressure. Citrate anticoagulant	Blood pressure decline beginning 40 minutes after trauma, and reaching 0, 40 minutes later. Survival, 1⅓ hours	Multiple fractures, femur	0.35

*Other than findings occurring regularly in shock.
†Estimated from weight differences of the hind quarters.

and the lower quarters were separated and weighed as described by Blalock (2).

The experiments were divided into two general groups. In the first, which served more or less as an orientation for the second, the course of the intact animal subjected to trauma to a hind limb was studied under various conditions. The limb was struck a number of times with a heavy round iron bar, breaking one or more bones. Breaks in the skin were carefully closed.

In the second group of experiments, the effect of trauma to a perfused hind limb separated from the body except for its nerve supply was studied. In 3 experiments, the circulation to the limb was isolated in a manner similar to the "anemic limb" preparation described by O'Shaughnessy and Slome (9). The limb was then perfused from the circulation of a heparinized donor cat, femoral artery and vein of the donor being joined to femoral artery and vein of the isolated limb by means of cannulas and rubber tubing. In 5 experiments the limb was perfused by means of a pump, the procedure being a modification of that described by Slome and O'Shaughnessy (12). An incision was made in the groin just large enough to permit the insertion of a dissecting scissors. The skin was separated without loss of blood from the subcutaneous tissues and cut along a line completely encircling the thigh at the level of the greater trochanter laterally, and the inferior margin of the symphysis pubis medially. The skin was then retracted and the subcutaneous tissues gathered into bundles by blunt dissection and cut between ligatures. The sciatic, femoral, and obturator nerves were next isolated, care being taken to preserve the vascular sheaths. The muscles of the thigh were then separated along the fascial planes for about an inch, the separation being carried as far centrally as possible, and two stout ties were placed around each muscle group about 1 inch apart. Branches of the femoral vessels arising between these two points were ligated. Muscle bundles not readily separated from the femur were included in ligatures around the bone. In this way the circulation to the limb was completely and surely isolated except for flow through the femur, which, in a number of experiments, was controlled by carefully stripping all muscle from the bone at the level of ligation, and by plugging the medullary canal of the proximal fragment of the femur with bone wax following the trauma. A postmortem examination was performed on the animal in each instance in order to be certain that damage had not been done to the nerves and that isolation of the circulation was complete.

The limb was perfused with blood drawn from a donor cat and rendered non-coagulable by the addition of 0.4 to 0.5 milligram chlorazol fast pink per cubic centimeter of blood (7). The perfusion system was much reduced in size so that the blood of one cat sufficed for each experiment. It included an oxygenator, filter, air trap, pressure by-pass, arterial manometer, warming coil and stromuhr. Thus blood could be supplied to the limb thoroughly oxygenated and at body temperature, the per-

TABLE II.—TRAUMATIC SHOCK IN THE INTACT CAT

No. Sex	Weight kgm.	Anesthesia	Procedure	Course	Adventitious pathology	Fluid loss in per cent body weight
15 M.	3.4	Chloralose 80 mgm. per kgm.	30 blows to thigh. Carotid pressure. Chlorazol fast pink anticoagulant	Blood pressure 125 and steady, 5½ hours after trauma. Animal sacrificed	Multiple fractures of femur	2.9
16 F.	2.9	Chloralose 80 mgm. per kgm.	30 blows to thigh. Carotid pressure. Chlorazol fast pink anticoagulant	Steady decline in pressure, reaching 0 in 3 hours	Multiple fractures of femur	3.6
17 M.	2.6	Chloralose 80 mgm. per kgm.	40 blows to thigh. 20 blows to leg, etc.	Steady decline in pressure, reaching 0 in 1½ hours.	Multiple fractures of femur as well as of tibia and fibula	6.1
18 M.	2.7	Chloralose 80 mgm. per kgm.	40 blows to thigh. 20 blows to leg, etc.	Steady decline in pressure, reaching 0 in 1¾ hours.	Multiple fractures of femur as well as of tibia and fibula	2.8

fusion pressure accurately adjusted, and the flow measured.

Other pertinent details of method and the sundry control procedures will be described together with the experimental results.

RESULTS

Experimental traumatic shock in intact cats. In 3 cats the femur was broken and the thigh muscles injured by 20 blows to the thigh. Nembutal was used as the anesthetic and citrate as the anticoagulant. The results of these experiments appear in Table I. It was noted in these and subsequent experiments in which citrate was used, that when a clot occurred in the cannula, the pressure following removal of the clot was always lower by 10 to 20 millimeters mercury, and recovered only slowly. In a number of instances the artery was clamped in the absence of clot, the tubing flushed, and the pressure set at the exact level at which it had been the moment previously. When blood was readmitted to the cannula the same fall was noted. Removal of 2 to 3 cubic centimeters of blood from the vein of a control animal (a quantity in excess of that entering the cannula when pressure in the tubing is closely approximated to pressure in the artery) produced no such effect, even though repeated a number of times. It was concluded, therefore, that citrate entering the artery was responsible for the fall in blood pressure after removal of a clot from the cannula. While it is well known that citrate lowers blood pressure, many investigators have nevertheless used citrate as the anticoagulant in the blood pressure recording system in their studies on shock, a practice which undoubtedly introduced an error into many such experiments.

In 4 animals, chloralose served as the anesthetic and chlorazol fast pink as the anticoagulant. This dye does not exert a depressor effect on blood pressure. Varying degrees of trauma were used, as is indicated in Table II, in which the results of these experiments are summarized. It was noted that more intense trauma was necessary to produce shock in the absence of citrate. The change in anesthetic could not be held accountable as will become clear later.

In 8 cats an effort was made to determine the effects of trauma when extremely light ether was used in the posttraumatic period. Four animals were lightly etherized and fixed to the animal board, by means of broad, soft restraints. In 1, a continuous blood pressure record was taken, citrate being used. In 3, blood pressures were recorded at the close of the experiment only. In all, ether and local anesthesia were used prior to cannulating the artery. The trauma consisted of 20 blows, as described, ether anesthesia being deepened during trauma to a point where there was no evidence of pain. It was greatly reduced promptly following trauma.

Shock rapidly ensued in the animal in which a continuous blood pressure record was taken. However, in the 3 remaining animals shock did not develop after many hours. Accord-

TABLE III.—TRAUMATIC SHOCK IN THE INTACT CAT

No. Sex	Weight kgm.	Anesthesia	Procedure	Course	Adventitious pathology	Fluid loss in per cent body weight
9 ..	1.9	Initial ether followed by ether analgesia	20 blows to thigh. Continuous carotid pressure. Citrate anticoagulant	Steady decline in blood pressure, reaching 0 in 3 hours	Multiple fractures of femur	1.1
10 F.	2	Initial ether followed by ether analgesia	20 blows to thigh. Terminal carotid pressure. Citrate anticoagulant	Blood pressure 135, 8 hours after trauma. Animal sacrificed	Multiple fractures of femur	...
12 F.	3.1	Initial ether followed by ether analgesia	20 blows to thigh. Terminal carotid pressure. Citrate anticoagulant	Blood pressure 90, 8 hours after trauma. Animal sacrificed	Multiple fractures of femur	2.3
13 F.	3.3	Initial ether followed by ether analgesia	20 blows to thigh. Terminal carotid pressure. Citrate anticoagulant	Blood pressure 110, 6 hours after trauma. Animal sacrificed	Multiple fractures of femur	3.4
8 ..	3.0	Initial ether followed by ether analgesia	20 blows to thigh	Alive 16 days later	...	...
20 F.	1.5	Initial ether followed by ether analgesia	30 blows to thigh. 20 blows to leg. 20 blows to foot. Terminal carotid pressure. Chlorazol fast pink anticoagulant	Blood pressure 75, after 8½ hours. Animal sacrificed	Multiple fractures of femur, tibia, fibula, and tarsal bones	6.0
14 M.	3.6	Initial ether followed by ether analgesia	30 blows to thigh. 30 blows to leg. 30 blows to foot. Terminal carotid pressure. Chlorazol fast pink, anticoagulant	Blood pressure 130, after 6 hours. Animal sacrificed	Multiple fractures of femur, tibia, fibula, and tarsal bones	5.1
19 M.	2.6	Initial ether followed by ether analgesia	30 blows to thigh. 30 blows to leg. 30 blows to foot. Terminal carotid pressure. Chlorazol fast pink anticoagulant	Blood pressure 45, after 8¼ hours. Animal sacrificed	Multiple fractures of femur, tibia, fibula, and tarsal bones	3.5

ingly, in the next 4 animals, more intense trauma was used. In the first, 20 blows were struck using greater force than previously, whereas in the others, both the number and force of the blows was increased.

Except for the first cat, which was permitted to survive, in each instance the carotid was cannulated under ether and local anesthesia before the experiment was discontinued and the blood pressure was measured after the effects of the ether had been dissipated. The animals were then sacrificed and fluid loss determined as described.

The first animal was alive and vigorous 16 days later. The 3 remaining were still alive after many hours, in spite of the intense trauma, and very extensive fluid loss. Only one was definitely in a state of shock, whereas in another, blood pressure was quite normal in spite of a fluid loss amounting to 5.1 per cent of the body weight.

The results of these 8 experiments are summarized in Table III.

It appeared from this first group of experiments that animals withstood trauma to a hind limb better when under very light rather than deep anesthesia in the posttraumatic period. It was also apparent that citrate affected the posttraumatic course adversely. Lastly it was concluded that 20 blows, as described by O'Shaughnessy and Slome (9), was not a severe enough trauma to insure uniform results in experiments of this type.

Experimental traumatic shock with only the nervous factor operative. In 3 instances, cross-circulation experiments were undertaken as

described. These proved to be very unsatisfactory, the donors dying before the recipients in each case. This method of study was therefore abandoned.

Control experiments. Ten animals were used in the perfusion experiments and controls. The control observations were made on 5 cats. In 4 animals under chloralose, the limbs were prepared as though for perfusion as described previously, the limb being denervated at the outset. Blood pressure was measured throughout this surgical procedure. In the first 2 animals, in spite of the fact that there was practically no blood loss, pressure declined markedly during the procedure, and displayed wide variations during this period. The total fall amounted to as much as 40 millimeters of mercury. However, a short period during which no manipulations were made permitted recovery.

In the first animal, when trauma was begun, it was rather surprising to note a sharp decline in blood pressure following the first several blows, since the limb had both its nerves and vascular connections with the body severed, and striking it should have been equivalent to striking the table. Accordingly, the remaining blows in this and the second animal were directed at the table, a very steep drop in pressure of as much as 45 millimeters of mercury resulting. This could be repeated, recovery always being quite prompt and maintained. Eight hours after the outset blood pressures were well above shock levels.[1]

In the third animal, spinal anesthesia was used in addition to the chloralose. Following the induction of spinal anesthesia, blood pressure declined from 120 to 80 millimeters of mercury. However, blood pressure remained quite stable throughout the surgical procedure of isolating the limb, showing none of the changes described in the first 2 control animals. With the preparation complete, blood pressure rose as the knee jerks returned, reaching its initial level, where it stood at the end of 5 hours.

In a fourth control animal the course was essentially as that described for the first 2, except that toward the close of the experiment the rectal temperature was inadvertently permitted to rise to 40 degrees C. Blood pressure dropped to 50, but with the removal of the heat it began to rise slowly. This trend was in evidence as the experiment was terminated 8 hours after the outset.

Lastly, the effect of severe jarring on blood pressure of the animal under chloralose, not operated upon, was studied. Under these circumstances, it was also found possible to produce marked depression, the blood pressure being driven from 150 down to 85 over a period of 1 hour and 20 minutes, the table being struck about 500 blows during this time. However, permanent depression could not be achieved by this means, the pressure eventually recovering. A refractoriness to this type of stimulus eventually became established, severe and prolonged jarring producing little or no effect. The results of these control experiments are summarized in Table IV.

Perfusion experiments. Five animals were used in the perfusion experiments. In 2 cats under chloralose anesthesia (80 milligrams per kilogram), a hind limb was isolated and perfused, as described, but the nerves were left intact. Over an hour after starting the perfusion, the flexion reflex, in each case, was equal on both sides. Trauma, consisting of 90 blows to the perfused limb, resulted in shock in the course of a few hours. Careful dissection, postmortem, showed the circulation of the perfused limb to be completely isolated in these and subsequent experiments.

In a third animal, 35 milligrams of chloralose per kilogram of body weight (reinforced with ether, as needed) was administered, less than half the customary dose. The animal was prepared for perfusion as described. Blood pressure stood at 145 at the time perfusion was begun. During the hour that followed certain adjustments were made that necessitated considerable manipulation of the wound. No blood loss was involved. The blood pressure fell slowly to 50 by the end of the hour. In the next hour the animal was undisturbed. Blood pressure returned to 115. Two and one-half hours after the beginning of perfusion, the flexion reflexes in both limbs were equal, and blood pressure was 115. The perfused limb showed no grossly visible edema.

[1]Later observations indicate that the fall of blood pressure obtained as a result of striking the cat board is accompanied by a sharp increase in ear temperature, suggesting a vasodilatation.

TABLE IV.—TRAUMATIC SHOCK IN THE ISOLATED INNERVATED LIMB PREPARATION. CONTROLS

No. Sex	Weight kgm.	Anesthesia	Procedure	Course	Adventitious Pathology
C1 M.	1.75	Chloralose 80 mgm. per kgm.	Limb isolated and denervated, carotid pressure. Chlorazol fast pink anticoagulant	Initial pressure, 130. Drop during procedure to 90. No blood loss. Recovery to 115 within an hour. Manipulation of proximal skin edge followed by fall of 20 mm. Hg. 100 blows to table accompanied by drop from 110 to 65 with prompt recovery. Same response elicited repeatedly. Pressure, 100, 8 hours after outset. Animal sacrificed	None
C2 F.	2.2	Chloralose 80 mgm. per kgm.	Limb isolated and denervated, carotid pressure. Chlorazol fast pink anticoagulant	Initial pressure, 115. Drop during procedure to 70. No blood loss. Rise to 125 within an hour. Manipulation of wound followed by fall from 110 to 60. Recovery to 95. 130 blows to table accompanied by drop from 95 to 65. Prompt recovery. Pressure 100, 8¼ hours after outset. Animal sacrificed	None
C3 F.	2.4	Chloralose and spinal	Limb isolated and denervated, carotid pressure. Chlorazol fast pink anticoagulant	Initial pressure, 120. Spinal anesthesia followed by fall to 80. Pressure steady at 80 throughout procedure. Knee jerks absent. Recovery to 120 in an hour. Knee jerks returned. Pressure 120, 5 hours after outset. Animal sacrificed	None
C4 F.	2	Chloralose 80 mgm. per kgm.	Limb isolated and denervated, carotid pressure. Chlorazol fast pink anticoagulant	Initial pressure recorded ½ hour following completion of preparation, 140. Manipulation of wound followed by fall to 60. Pressure 125, 5½ hours after outset. Rectal temperature inadvertently permitted to reach 40°C. Pressure fell to 50. Pressure slowly recovering when experiment terminated, 8 hours after outset	None
C5 M.	2.3	Chloralose 80 mgm. per kgm.	Carotid pressure. Chlorazol fast pink anticoagulant. Indirect trauma	Initial pressure, 150. 500 blows struck to table over period of 1½ hours. Pressure, 85. Recovery to 110 in an hour. 500 blows in next hour without effect	None

Five hours had elapsed since the administration of the anesthetic. The pupils were markedly constricted. Ether anesthesia was induced and 90 blows were delivered to the limb. The broken femur was exposed at once and the medullary canal was plugged with bone wax. A drop in pressure of about 5 millimeters of mercury was noted during trauma, followed by recovery to the initial value of 115. The marked depressor response to jarring of the cat board seen in the control animals under chloralose anesthesia was absent. The ether was greatly reduced after trauma, and shortly thereafter the pupils were again slit-like. Two and one-half hours after trauma and 6½ hours after the first dose of chloralose, the blood pressure was still 115, and the animal was still under ether analgesia. Chloralose to the amount of 70 milligrams per kilogram of body weight was injected intravenously at this time. A few minutes later, the animal board was struck several times and this sent the blood pressure from 115 to 70, but prompt recovery followed. Within 20 minutes of the second dose of anesthesia the blood pressure had fallen 25 millimeters; within 65 minutes, it had declined to 0.

Two animals were prepared for limb perfusions under spinal anesthesia, reinforced with light ether as needed. Before observations were begun, all evidence of the spinal anesthesia had disappeared, and flexion reflexes on both sides were equal. Ether was given during trauma, which consisted of 90 blows to the isolated, innervated, perfused limb. In neither animal was the marked depressor response to jarring seen. The first cat was rather deeply etherized previous to trauma. Its pressure fell from an initial value of 110 to 85 with anesthetization. During trauma there was a momentary rise of pressure to 110, followed by a prompt decline to 85. The broken femur was plugged proximally with bone wax and the ether discontinued, to be renewed only in analgesic quantities. Blood pressure slowly rose, reaching its initial level within 2 hours after trauma, and remaining there for about an hour, after which it slowly declined, reaching 0 in 5¼ hours after trauma. In the second animal, trauma was not begun until 3¼ hours after starting the perfusion. There was no evidence of edema in the experimental limb, its nervous responses equaling those of the normal side. Ether was administered. The blood pressure varied little from its initial value of 115. With trauma there was a drop of 10 millimeters of mercury, with immediate recovery. The femur

TABLE V.—TRAUMATIC SHOCK IN THE ISOLATED INNERVATED LIMB PREPARATION

No. Sex	Weight kgm.	Anesthesia	Procedure	Course	Adventitious Pathology
P3 M.	2.5	Chloralose 80 mgm. per kgm.	Limb isolated and perfused. Nerves intact. 30 blows to thigh, 30 blows to leg. 30 blows to foot, carotid pressure. Chlorazol fast pink anticoagulant	Initial blood pressure, 140. Pressure to 80 during trauma. Prompt recovery to 100 following trauma. Steady decline to 0 in 2½ hours	Multiple fractures of femur, tibia, fibula, carpal bones
P4 F.	2.3	Chloralose 80 mgm. per kgm.	Limb isolated and perfused. Nerves intact. 30 blows to thigh, 30 blows to leg, 30 blows to foot, carotid pressure. Chlorazol fast pink anticoagulant	Initial blood pressure, 100. Pressure to 60 during trauma. Prompt recovery to 85 following trauma. Steady decline to 0 in 3¼ hours	Multiple fractures of femur, tibia, fibula, carpal bones
P12 F.	2.2	Chloralose 35 mgm. per kgm	Limb isolated and perfused. Nerves intact. 30 blows to thigh, 30 blows to leg, 30 blows to foot, carotid pressure. Chlorazol fast pink anticoagulant	Initial pressure, 115. Recovery from initial anesthetic. Ether. No change in blood pressure. Trauma. Pressure to 110 during trauma. Ether analgesia henceforth. Pressure 105, 2½ hours after trauma. Chloralose, 70 mgm. per kgm., given. Prompt decline to 0 in 1 hour	Multiple fractures of femur, tibia, fibula, carpal bones
P13 M.	2.4	Spinal and ether	Limb isolated and perfused. Nerves intact. 30 blows to thigh, 30 blows to leg, 30 blows to foot, carotid pressure. Chlorazol fast pink anticoagulant	Initial pressure, 110. Ether. Pressure to 85. Trauma. (Ether analgesia henceforth). Pressure, 85. Recovery to 100 within 1 hour. Maintained above 100 for 2 hours. Slow fall to 0 in the next 2¼ hours. Survival, 5¼ hours	Multiple fractures of femur, tibia, fibula, carpal bones
P14 F.	2.5	Spinal and ether	Limb isolated and perfused. Nerves intact. 30 blows to thigh, 30 blows to leg, 30 blows to foot, carotid pressure. Chlorazol fast pink anticoagulant	Initial pressure, 115. Ether. No change in pressure. Trauma. (Ether analgesia henceforth). Pressure, 110. Pressure, 120, 5¼ hours after trauma. Animal sacrificed	Multiple fractures of femur, tibia, fibula, carpal bones

was plugged promptly. Five and one-fourth hours later, when the experiment was terminated, the arterial pressure was 120.

In these 2 animals it was noted that periods of excitement under ether analgesia almost uniformly caused a fall rather than a rise in blood pressure. The results of the perfusion experiments are summarized in Table V.

ANALYSIS OF STUDY

The argument advanced against the nervous factor in traumatic shock has been based entirely on evidence of a negative character. (Blalock, 2; Parsons and Phemister, Cannon, Freedlander and Lenhart, and others). This has consisted largely of experiments in which peripheral nerves or central pathways have been interrupted without influencing the onset of shock. In other experiments peripheral nerves have been stimulated faradically without the production of shock. In still others, the magnitude of fluid loss has been accepted as sufficient proof that the nervous factor was of no consequence. In none of these experiments were fluid loss ruled out and the rôle of the nervous system studied independently.

In those investigations of Slome and O'Shaughnessy (12); O'Shaughnessy and Slome (9); Bell, Clark, and Cuthbertson (1); and Blalock and Cressman (3) in which an effort was made to study the nervous factor under controlled conditions, the lack of agreement apparently revolves around a number of points of procedure. Only the cross-circulation and not the mechanical perfusion experiments of O'Shaughnessy and Slome were repeated, in spite of the fact that the former employ an extremely difficult and unreliable

method. In addition, the degree of trauma used in these experiments was apparently not intense enough invariably to give a clear-cut result. Differences in the anesthesia used probably also accounted, in part, for variations. It should be noted, however, that the abrupt terminal drops in blood pressure, and deaths apparently due to respiratory failure described by Blalock and Cressman (3) in experiments in which chloralose was used, were not encountered in the present study. No untoward effects ascribable to this anesthetic were seen at any time.

In the studies here recorded, an effort was made to obviate these criticisms. Trauma in many experiments was quite intense. In several experiments, anesthesia was withheld in so far as permissible. The use of citrate for continuous blood pressure recording was avoided. And finally, the perfusion experiments were done by means of a very accurate and dependable mechanical system. In addition, the possible loss of blood through the fractured femur, a factor not controlled by O'Shaughnessy and Slome, was checked in several experiments. When all these precautions were observed, it was still possible to produce fatal shock by traumatizing a limb having only its nervous connections with the body intact.

In attempting to integrate the nervous and fluid-loss factors, Slome and O'Shaughnessy (12) ascribed the initial depressor effect following trauma to a cat's limb to fluid loss, and the subsequent fall in blood pressure, to nervous impulses arising in the traumatized area. However, a marked initial depression of blood pressure was always seen in the present studies, even though the limb was being perfused and there was no fluid loss, provided the animal was under chloralose anesthesia. As has been described, striking the table was quite as effective as striking the limb, in this respect, and such an effect could certainly not have been mediated except through the nervous system. However, since this last observation could not be reproduced in animals under ether analgesia, too much weight cannot be attached to it.

The conclusion drawn by O'Shaughnessy and Slome (9), that anesthesia serves to protect against shock by blocking so called nociceptive impulses arising from the traumatized area, cannot be substantiated. Though such impulses may actually be depressed, general anesthesia apparently impairs other functions sufficiently that the balance is tipped toward the other side. Venous return, mediated by muscular activity, may be one factor involved. In addition, Seevers and Stormont have shown that general anesthesia in both man and animals impairs the ability to adjust to changes in blood ion concentration. In this connection it is of interest that Freeman and his co-workers reported an acidosis in animals going into shock following hemorrhage.

Although the argument throughout has concerned itself solely with that type of shock produced in the experimental animal by trauma to an extremity, the observations of Taylor, Weld, and Harrison are of interest as they bear upon the concept of a nervous mechanism in shock, generally. These investigators reported the production of shock in dogs by distending segments of bowel, without obstruction. This was accomplished by the insertion of a distensible balloon attached to a heavy piece of rubber tubing into the bowel, which was then distended by inflation of the balloon. Continuity of the intestinal passage was maintained by the tube. Shock could be prevented by the denervation of the length of bowel prior to its distention.

CONCLUSIONS

1. Shock can be produced in the cat by appropriate trauma to a perfused hind limb separated completely from the general circulation, and communicating with the body only by means of its nerves. This can be demonstrated in the animal under chloralose anesthesia, as well as in the animal under ether analgesia in the posttraumatic period.

2. The animal under ether analgesia appears to be more resistant to shock produced by trauma to an artificially perfused innervated limb than is the more deeply anesthetized animal (under chloralose).

3. The intact animal, under ether analgesia in the posttraumatic period, is much more resistant to shock produced by trauma to an

extremity than is the intact animal under chloralose or nembutal anesthesia. The animal under ether analgesia is thus more resistant to the nervous and fluid loss factors combined, in experimental traumatic shock, than is the anesthetized animal.

The experiments here reported have been designed to test the reality of a nervous factor in traumatic shock and shed no light whatever upon the relative importance of this as compared with other causative factors.

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CLINICAL SURGERY

A NEW VALVULAR CHOLECYSTOGASTROSTOMY

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FOLLOWING a direct anastomosis of the gall bladder to the stomach, the biliary system is no longer protected as it was normally by the valve-like action of the sphincter of Oddi and the constricting action of the duodenal musculature.[1] The direct interchange of contents occurring between the gastro-intestinal tract and the gall bladder may predispose to infection of the biliary tract varying from mild to very severe toxic states. Even though such extension of the inflammation may not always manifest itself clinically, there are surgeons who state that cholangitis and hepatitis invariably occur after cholecystogastrostomy. Those who have observed and reported these post-operative conditions have devised methods which were to prevent them, but which were not as successful as expected. Whipple found cholangitis and hepatitis resulting from cholecystogastrostomy when this operation was combined with gastro-enterostomy preliminary to resection of the head of the pancreas. Lahey and McKinnon advocated the use of the upper portion of the jejunum for anastomosis with the gall bladder in an effort to eliminate an ascending infection of the biliary tract. To avoid a direct interchange of contents between gall bladder and jejunum, Whipple employed the type of direct cholecysto-jejunostomy originally suggested by Monprofit. Brunschwig and others employed a long loop of jejunum for cholecystenterostomy combining it with an entero-enterostomy as an additional safeguard. Wangensteen is of the opinion that the site between the gastro-intestinal tract and the gall bladder is not as important as the size of the opening which is to be adequate for drainage.

In an x-ray study of patients with cholecysto-gastrostomies, Bernhard found in at least 50 per cent of the cases a regurgitation of the barium into the gall bladder and biliary passages. Fogelson and Bachrach reported similar findings in dogs with cholecystogastrostomies, but stated that they could not attribute the periportal hepatitis which they saw to an ascending infection. They further noted that hypo-acidity and infection of the common duct co-existed in these animals. Zollinger experimented with two groups of dogs in which he employed two methods of cholecystogastrostomy. In one group he followed the ordinary procedure; in the second group he performed his original valvular cholecysto-gastrostomy. At autopsy he found food particles and hair in the gall bladder and the biliary tracts only in the animals which had been subjected to ordinary cholecystogastrostomy, but none whatsoever in the second group. In a further examination of the biliary tracts in both groups after sodium iodide injections, Zollinger observed that the intrabiliary and extrabiliary tracts were grossly dilated only in the cholecystogastrostomies which did not include a valve; also, when air was injected into the stomach its prompt entrance into the biliary tree indicated again the ease with which bacteria-laden food particles may make their way into the gall bladder and biliary tract.

Turning now to actual methods of anastomosis between stomach and gall bladder which have been devised to prevent the postoperative sequelæ mentioned, it was Mason who first developed a procedure with that objective in mind. After making a tunnel between the submucosa and muscularis of the anterior stomach wall, in the region of the antrum from lesser toward greater curvature, he drew the fundus of the gall bladder through it, and established the anastomosis with the gastric lumen. Spivack's method of valvular cholecystogastrostomy is similar to that of Mason, except that he employs a tunnel made in a horizontal direction on the antral wall. Mirrizzi modified Mason's method by establishing the tunnel in the duodenal wall.

Gentile applying Coffey's principle of transplanting the ureter, which depends upon the formation of a stoma by the sloughing of a transfixed suture between ureter and colon, failed to produce by this method a successful anastomosis between gall bladder and stomach in his experi-

[1]The technique herein described was perfected in the Department of Anatomy of the University of Illinois, College of Medicine, with the aid of Dr. Otto F. Kampmeier, professor of Anatomy and head of department. The writer wishes to express his indebtedness to Dr. Kampmeier for his invaluable aid.

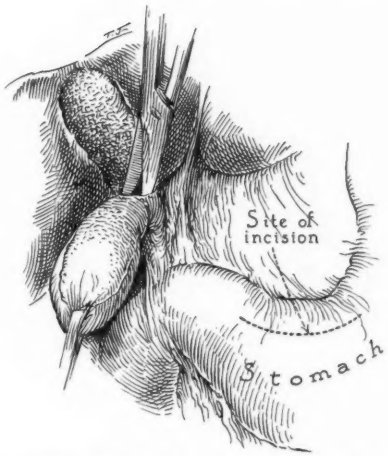

Fig. 1. The gall bladder is mobilized from its liver bed by dissection. The dotted line on the anterior surface of the stomach antrum indicates the site of incision and anastomosis.

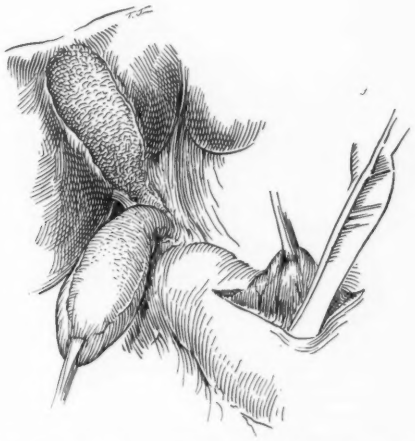

Fig. 2. The seromuscular incision is made, and the herniation of gastric mucosa is increased by traction upward with an Allis forceps and by blunt stripping.

mental animals. Zollinger repeatedly stresses the fact that in order to assure a patent stoma such an opening between gall bladder and stomach must actually be established at the time of operation. Zollinger's own method of valvular cholecystogastrostomy consists of making a seromuscular incision into the stomach from lesser to greater curvature, then fixing and anastomosing the gall bladder to the stomach near the greater curvature and sewing the edges of the seromuscular gap over the body of the gall bladder by interrupted sutures. The valve-like effect of the confluence produced by this method depends upon the formation of a flap, consisting of gall bladder wall and gastric mucosa, which, when pressed against the anterior gastric wall by food entering the stomach, closes the stoma leading into the gall bladder.

Experimenting with a new mode of attack, the present writer devised a valvular cholecystogastrostomy which appears more simple and is an improvement over the preceding methods. It represents a circular or teat-like valve formed at the junction of the gall bladder and stomach which is successful in preventing the gastric contents from regurgitating into the biliary tract. The procedure of establishing such a valve is as follows:

Through either a transverse, or a Kocher type of incision of the abdominal wall, the gall bladder and stomach are exposed. The gall bladder, being usually distended, easily presents itself in the wound. The common, hepatic and pancreatic ducts are examined, and then the character of the contents of the gall bladder determined by aspiration. The presence of normal or turbid bile within the gall bladder indicates that the bile is passing freely into it, and that the anastomosis should produce a favorable result. However, when limpid white bile is withdrawn into the syringe, surgery is not indicated, for if done it is worse than useless since it is followed by a high mortality.

The body of the gall bladder in most instances has to be dissected from its liver bed sufficiently to permit the fundus to be brought down to the anterior surface of the gastric antrum.

A slightly curved seromuscular incision about 5 centimeters long is made on the anterior surface of the antrum, parallel to and approximately 2 centimeters from the lesser curvature, as shown in Figure 1. This step permits the gastric mucosa to bulge out far enough to enable it to be grasped and pulled upward by means of an Allis forceps (Fig. 2). By continued traction and careful blunt stripping at the line of submucosal cleavage between the muscularis and mucosa (Fig. 2), a greater herniation of the gastric mucosa occurs. A mucosal outpouching of approximately 3 to 4 centimeters is considered sufficiently long to proceed with its attachment to the gall-bladder fundus. Sharp dissection should be avoided wherever possible in order not to cut through the mucosa. (Incidentally, this pro-

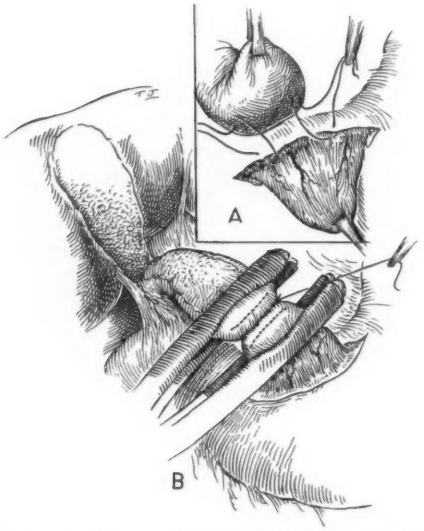

Fig. 3. A, Sutures are inserted into the seromuscular coat of the posterior wall of the gall bladder about 2 to 3 centimeters from the fundus, continued through the same layers of the posterior lip of the stomach incision and temporarily left untied. B, A serosal stitch has been completed between gall-bladder fundus and apex of the herniated gastric mucosa. The dotted lines represent sites of incision for opening and anastomosing the two organs.

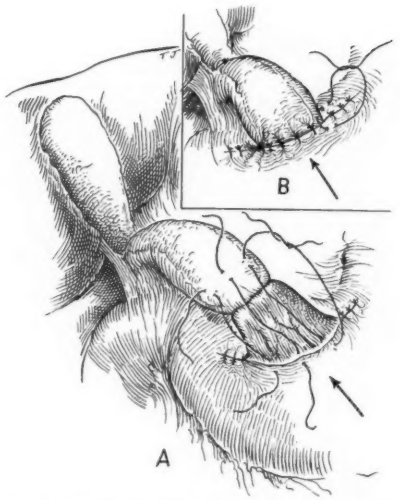

Fig. 4. A, The anastomosis is completed between the gall bladder and gastric mucosa. Interrupted sutures, like those that were left untied on the posterior aspect of the anastomosis, are now placed on the anterior aspect of the junction of the two organs. B, The posterior and anterior interrupted sutures have been tensed and tied, completing the invagination of 2 to 3 centimeters of gall-bladder fundus and about 4 to 5 centimeters of gastric mucosa into the lumen of the stomach. The arrows indicate the plane of section represented by diagrammatic sketches in Figure 5.

cedure of causing a herniation of the gastric mucosa should solve the problem of approximating the biliary system and the stomach in those few cases of cholecystogastrostomy, or those requiring choledochogastrostomy, in which the stomach cannot be mobilized easily; this procedure would, of course, necessitate peritonealizing the drawn out mucosal anastomosis.)

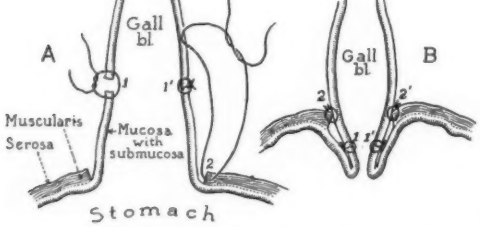

Fig. 5. A, Diagram of section taken at plane represented by the arrows in Figure 4, i.e., in the longitudinal axis of the anastomosis. *1*, An untied interrupted suture connecting all layers of the gall-bladder wall to gastric mucosa and adherent submucosa. *2*, Interrupted suture not tied, to illustrate its passage from a point in the wall of the gall bladder 2 to 3 centimeters above its fundus to and through a point in the seromuscular layer at the edge of the gastric incision. B, Illustrates sutures *1—1'* and *2—2'* tensed and tied, and hence the completion of the invagination with its resultant valve-like formation.

Before anastomosing the mucosal outpouching of the stomach with the fundus of the gall bladder, 4 to 6 interrupted sutures are passed through the seromuscular layer of the posterior wall of the gall bladder at a point about 2.5 centimeters from the tip of the fundus and then through the seromuscular layer at the posterior edge of the stomach incision (Fig. 3, A). These posteriorly placed sutures are kept on forceps and are not tied until the anastomosis is completed. Both the fundus of the gall bladder and the apex of the protruding gastric mucosa are compressed with intestinal clamps, approximated, and stitched together by a continuous serosubmucosal suture, as shown in Figure 3, B. Each organ is then opened for a distance of 2 centimeters, as indicated by the dotted lines in the same figure. The anastomosis is completed by following the usual procedure.

Four to 6 sutures are now placed anteriorly in exactly the same manner as those placed posteriorly, as illustrated in Figure 4, A. First the posterior interrupted sutures are tensed and tied and then the anterior ones (Fig. 4, B), causing the entire process of gastric mucosa and gallbladder fundus to invaginate into the lumen of the stomach, as depicted diagrammatically in Figure 5.

To close the remaining ends of the incision originally made to produce the maximum of mucosal herniation, interrupted sutures are employed, as shown in Figure 4, B. The same figure also illustrates two corner sutures which have the purpose of further inverting and narrowing the lumen of the anastomosis, and thereby puckering the circular fold of the valve for greater efficiency. A piece of omental tissue may be applied externally to the anastomotic junction as an additional precaution against biliary leakage.

Diagrams A and B of Figure 5 represent sections taken through the longitudinal axis of the anastomosis, that is, in the direction of the arrows in Figure 4, and illustrate the mode of production of the valve. Points *1* and *1′* in A indicate respectively the two steps in placing the sutures and joining the opened gall bladder and gastric mucosal diverticulum. Suture *2*, placed but not yet tensed in the same diagram, shows its course from a point in the wall of the bladder, about 2 to 2.5 centimeters above the anastomotic junction, to and through the seromuscular layers at the edge of the incision in the stomach. Diagram B of Figure 5 depicts the invagination completed by the tensing and tying of all sutures *2*, with the resultant formation of the circular valve.

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THE SURGICAL TREATMENT OF UNEQUAL LEG LENGTH

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INEQUALITY of leg length is a frequent cause of unsightly gait which must always be remembered in analyzing disability in the lower extremities. Frequently the causative agent or disease has long since disappeared or has been brought under control, the inequality in leg length remaining as the important residual disability. In previous decades the orthopedic surgeon was content to prescribe a raised shoe or other appliances, but in recent years the comparative safety of aseptic bone surgery and the development of good technical methods of shortening or of elongating the long bones have caused such palliative measures to become antiquated except when indicated for minor discrepancies of leg length. Most leg length discrepancies of 1 to 4 or 5 inches can be eliminated by an appropriate surgical procedure. Such methods should be applied as early as possible in life to avoid personality maladjustments and are rarely indicated after the twenty-fifth year. A review of the causes of unequal leg length is presented in Table I. Infections, poliomyelitis, and trauma are most commonly encountered.

PREVENTION OF SHORTENING

Correct treatment of the conditions mentioned inaugurated at the earliest possible date will do much to reduce the incidence of inequality of leg length. Early attention to tuberculosis and to pyogenic osteomyelitis will avoid perforation of the infectious process through the epiphyseal cartilage and into joints, two of the major mechanisms producing unequal limb length in these conditions. Surgical treatment of tuberculous joints at the appropriate time is to be preferred to the purely conservative management of these infections, as in many instances atrophy is promoted and longitudinal growth retarded by prolonged immobilization in a cast. Unequal involvement of the epiphyseal growth cartilage with resultant deformity as well as shortening by enchondromas, gummas, or infections can be combated by repeated observation and appropriate operative closure of the growing side of the epiphyseal line. Unequal limbs, the sequellæ of poliomyelitis, are rare in cases from the more recent epidemics as prolonged immobilization has not been prescribed often, and patients are now encouraged to walk suitably braced within a few weeks or months after the onset of the disease. The early recognition and treatment of tumors and fibrocystic and suppurative bone disease will avoid pathological fracture which is at times complicated by shortening.

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PHYSIOLOGY OF BONE BEARING UPON LONGITUDINAL GROWTH DISTURBANCE

Closure of epiphyseal lines by trauma associated with fractures has been reported by Compere (14) and others. In fractures of the shaft of the femur in young children with shortening the result of overriding, there may be equalization of limb length by compensatory overgrowth at the metaphyses of the bones of the shortened leg, as shown by David. However, the older the child, the less the amount of compensation. A difference of leg length up to 1 inch or slightly more may be concealed by tilting the pelvis.

Involvement of the epiphyses about the knee is likely to contribute more shortening than that at the hip or about the ankle, since the studies of Digby, Bisgard (8) and others (46) indicate that approximately 12, 40, 28, and 20 per cent of the growth of long bones in the lower extremity occurs at the upper femoral, lower femoral, upper tibial and lower tibial epiphyses respectively. From a practical point of view, operative fusion of the hip in very young children when the epiphyseal line is of necessity damaged or bridged by bone grafts, results in very little or no shortening unless there is prolonged cast immobilization (26). The same thesis has been experimentally demonstrated by Compere and Garrison (16) and by Harmon and Adams in children in whom the capital femoral epiphysis has been destroyed by suppuration. In the latter case it is not certain if the mechanism operating is that of overgrowth at the epiphyses on the diseased side, the side operated upon, or compensatory slowing of growth in the epiphyses of the sound limb.

Elongation of bone produced by abnormal vascularity or by disease in the region of the metaphysis is occasionally observed, but this is not as common as is shortening due to disease. Horton

TABLE I.—THE CAUSES OF INEQUALITY OF LEG LENGTH

1. Fractures
   a. with overriding
   b. into the epiphyseal line creating growth arrest
2. Bone infections
   a. pyogenic osteomyelitis
   b. tuberculosis
   c. variola
   d. syphilis
3. Bone tumors
   a. enchondromas
   b. exostoses
   c. fibrocystic disease
   d. neurofibromatosis
4. Congenital abnormalities
   a. congenital absence or malformation of bones
   b. arteriovenous aneurisms
   c. hypertrophy, regional or hemihypertrophy
5. Disuse retardation of growth
   a. residual of poliomyelitis
   b. prolonged cast immobilization in growing children.

Conditions 1 and 2 produce shortening by loss of bone or by premature closure of the epiphyseal line.

and others have reported cases in children in which arteriovenous aneurisms caused lengthening of bones on the diseased side. Pyogenic foci in the metaphyses, especially if untreated, may stimulate a bone to grow excessively. Most of such overgrowth is either of relatively small extent or is compensated by growth in later years. For a long time it was believed that unilateral lumbar sympathetic ganglionectomy or ramisectomy would cause stimulation in growth of the homolateral limb as when atrophied from the aftereffects of poliomyelitis. However, it has now been shown experimentally by Bisgard (7) and by careful teleroentgenographic measurements in growing boys by Fahey that such operations have no consistent effect on the longitudinal growth of an extremity. This question needs restudy under carefully controlled conditions which will utilize more careful measurements.

OPERATIVE SLOWING OF GROWTH OF THE LEG

Phemister was the first to advise that operative arrestment of longitudinal growth be applied to the bones of the longer extremity and described the operative technique that would result in cancellation of epiphyseal growth. This procedure as originally described involves sliding small bone grafts cut from the shaft contiguous to the epiphyseal line across the structure and gouging out the cartilage disc to a depth of 1 to 2 centimeters along the sides (Fig. 1). Thorough and equal disruption of the line of enchondral ossification in the juxta-epiphyseal zone is necessary, since in two of the cases in this series there was evidence

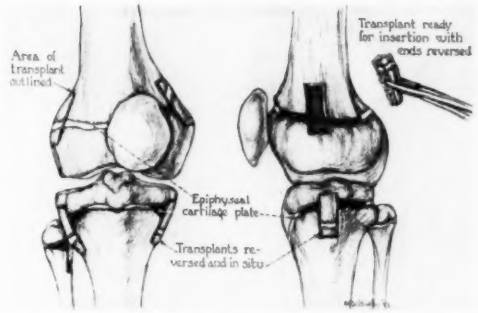
Area of transplant outlined
Transplant ready for insertion with ends reversed
Epiphyseal cartilage plate
Transplants reversed and in situ

Fig. 1. Technique of epiphyseal growth arrest operation —Epiphyseodesis as described by Phemister. (Courtesy of Dr. D. B. Phemister and the *Journal of Bone and Joint Surgery.*)

of unequal cancellation of growth on the two sides with deformity. Campbell also reported a case of genu recurvatum caused by unequal operative blockage of the anterior portion of the epiphyseal line of the tibia. This method is applicable for equalization of leg length only during the latter half of the growing period. *It has a definite field of usefulness, but the greatest care must be practiced in the selection of cases for the operation, especially in the selection of the appropriate age for a given amount of shortening.* There is no mathematical formula for obtaining an exact amount of leg equalization by this method. Even when operation is performed under proper indications, the results are occasionally uncertain. Other factors such as the number of epiphyses to be closed and the age at which the operation is to be done should be carefully evaluated. The data given by Hatcher are of extreme value in calculating the probable shortening to be obtained at various ages, since his measurements were obtained by teleroentgenograms. The variability of arrest of longitudinal growth is well illustrated by his material. The usual mistake in this series was

TABLE II.—AVERAGE NORMAL GROWTH TO BE EXPECTED FROM LOWER EXTREMITY
(Calculated from data of Baldwin)

Age in years	Male cm.	Female cm.
7	28.88	21.05
8	26.47	18.39
9	23.26	16.02
10	20.14	12.16
11	17.07	8.37
12	14.34	6.40
13	10.70	3.51
14	7.27	1.80
15	4.24	0.52
16	2.09	0.14

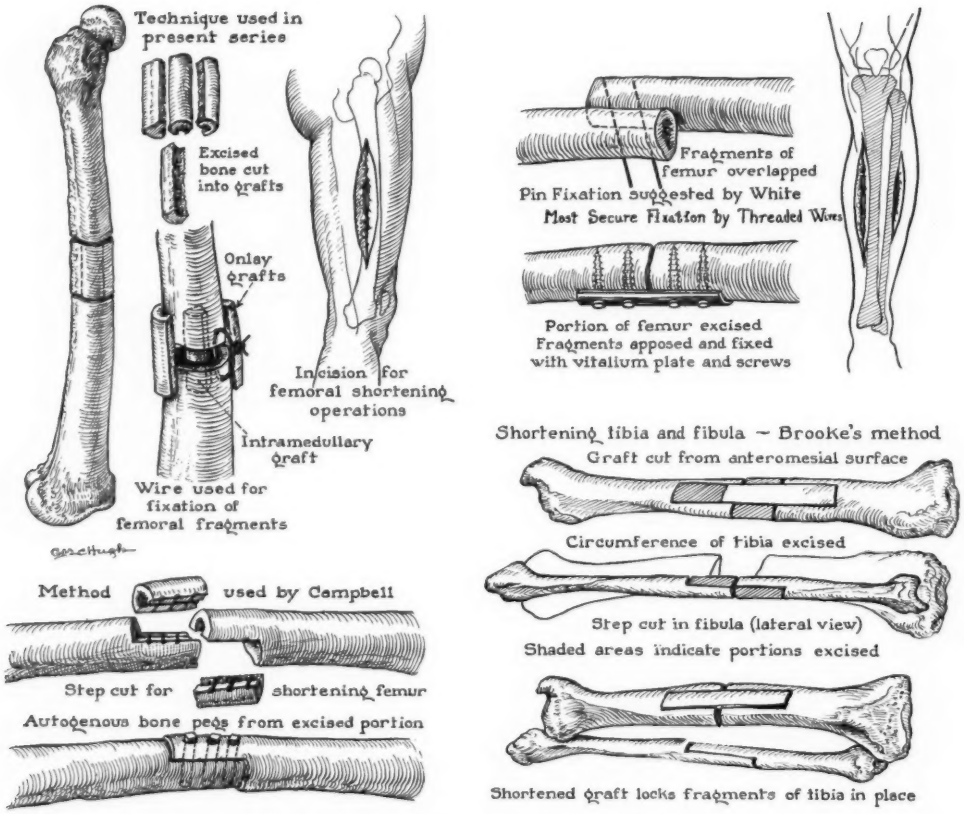
Technique used in present series
Excised bone cut into grafts
Onlay grafts
Incision for femoral shortening operations
Intramedullary graft
Wire used for fixation of femoral fragments
Method used by Campbell
Step cut for shortening femur
Autogenous bone pegs from excised portion
Fragments of femur overlapped
Pin Fixation suggested by White
Most Secure Fixation by Threaded Wires
Portion of femur excised
Fragments apposed and fixed with vitallium plate and screws
Shortening tibia and fibula ~ Brooke's method
Graft cut from anteromesial surface
Circumference of tibia excised
Step cut in fibula (lateral view)
Shaded areas indicate portions excised
Shortened graft locks fragments of tibia in place

Fig. 2. Methods of shortening the bones of the longer sound extremity. The method of resection, with the use of intramedullary and onlay grafts was employed in the majority of cases of this series.

failure to operate at a sufficiently early age and to arrest enough epiphyses. Data should be available upon racial and familial growth expectancy, the total leg length of the patient and the length of each bone. Cognizance should be taken of sex, since longitudinal growth ceases from 2 to 4 years earlier in girls than in boys (6, 23, 28). This information can be obtained in the usual way by external mensuration, but can be obtained more accurately by stereoteleroentgenograms of the extremities. The approximations based upon the work of Baldwin are given in Table II. The most accurate measurements of growth increments for each bone at various ages in children are available in the article by Hatcher. For 3 inches shortening at the age of 8 in girls and 11 in boys it is generally necessary to arrest more than one epiphysis, usually the two at the knee, since they together contribute more than 70 per cent of the total limb growth. Prior to these ages for this or greater shortening it is advisable to arrest only the lower femoral epiphysis or a tibial and fibular epiphysis at one end. The patient should be observed carefully and other epiphyses closed as soon as it is determined that the original closure is not being effective in equalizing the limbs. These general indications should be modified in light of the accessory data for age, sex, and leg discrepancy given in Table II. For example, members of the southern European races in whom short stature is the rule should be operated upon at a relatively early date. Past the age of 11 or 12 in girls and 13 to 15 in boys, this operation should rarely be done, as very little or no longitudinal growth can then be expected in the long bones. Roentgenograms of the ends of the bones should always be ob-

tained prior to operation after the age of 9 in girls and 12 in boys to be certain that premature closure of the epiphyseal line has not occurred. In over three thousand miscellaneous orthopedic cases under the age of 21 years, seen by one of the authors during the past 2 years, the growth arrest operation was recommended only eight times. In 43 cases of predominatingly unilateral acute poliomyelitis, followed for the past 5 years by us, none has developed enough shortening to warrant a limb equalization of any kind. Of these cases, 27 patients passed through the pre-adolescent growth period during this time.

EQUALIZATION OF LEG LENGTH BY SHORTENING THE LONG LEG

Shortening of the longer sound limb by excision of a portion of the diaphysis has been reported repeatedly in the surgical literature but has not become popular until the past few years. Rizzoli in 1847 is credited by Steindler as having performed the first leg shortening by cutting the bone and allowing the femoral fragments to override. Sayre was credited with priority (1863) by Brooke who used the same method. Cases have been reported by Mayer and Nussbaum, Glessner, Taylor, Fassett, Calvé and Galland, Shands, and others (11, 18, 30, 31, 34, 37, 38). White (45) and Brooke presented bibliographies, while Calvé and Galland and Brooke brought out and described methods that were sound, the latter being the first to describe an operative method of shortening the tibia.

These methods are all more simple than the methods of operative lengthening. In addition, the likelihood of disaster is slight in the shortening operation, while unfavorable complications are common during the lengthening procedure. This method of leg length equalization has an extensive field of application, since it is mathematically accurate when the more exact methods are employed. The risk is greater than in the epiphyseal arrest operation but not sufficiently so to prevent wide application. Either the tibia or femur may be shortened as much as three inches, but the femoral shortening operation is usually more applicable. The site of election for shortening is at the junction of the middle and lower thirds of the femur and in the middle third of the femur. Only the mathematically exact methods are described here, since shortening by allowing the osteotomy fragments to override is inexact and difficult to control.

The technique employed in shortening the femoral diaphysis in this series of cases is shown in Figure 2. The midshaft of the femur is approached

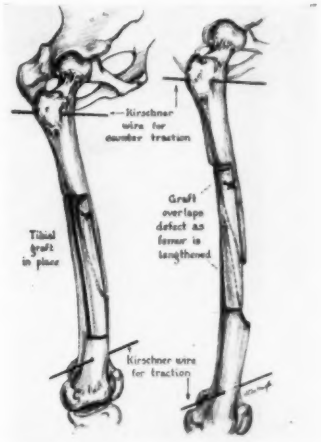
Kirschner wire for counter traction
Graft overlaps defect as femur is lengthened
Tibial graft in place
Kirschner wire for traction

Fig. 3. The technique of femoral bone lengthening by means of an autogenous tibial bone graft and skeletal traction. (Courtesy of Dr. E. L. Compere and the *Journal of Bone and Joint Surgery*.)

along the posterolateral intermuscular septum. The periosteum is incised and stripped only for the length of the proposed shortening. A Gigli saw is then used to sever the bone transversely at the upper limits of the proposed shortening which, in the femur, is usually at the junction of the middle and lower thirds. The distal fragment is angulated allowing its proximal end to protrude from the wound. The desired length of excess bone is removed by a hand saw. The excised bone is then split into several fragments, one of which should be of sufficient size to serve as a snugly fitting intramedullary graft. It should increase in diameter from above downward according to the width of the medullary canal. The remaining pieces of bone are used as onlay grafts to bridge the osteotomy site. Bronze-aluminum wire, which has been used in 7 cases in this series, as illustrated, aids in preventing separation of the bone ends. It is, however, not essential. An alternative technique, advocated by Campbell, is shown in the insert, and the method of tibial shortening described by Brooke is also shown. His accurate step cut operation on the fibula is not essential. Such modifications are technically more difficult and require wider periosteal stripping. They have the merit of preventing angulation by internal fixation. The same effect has been obtained by attaching an onlay graft to the lateral cortex with either autogenous bone pegs or with screws. Vitallium plates and screws have come into favor during recent years. Whether used for fixation or not, the

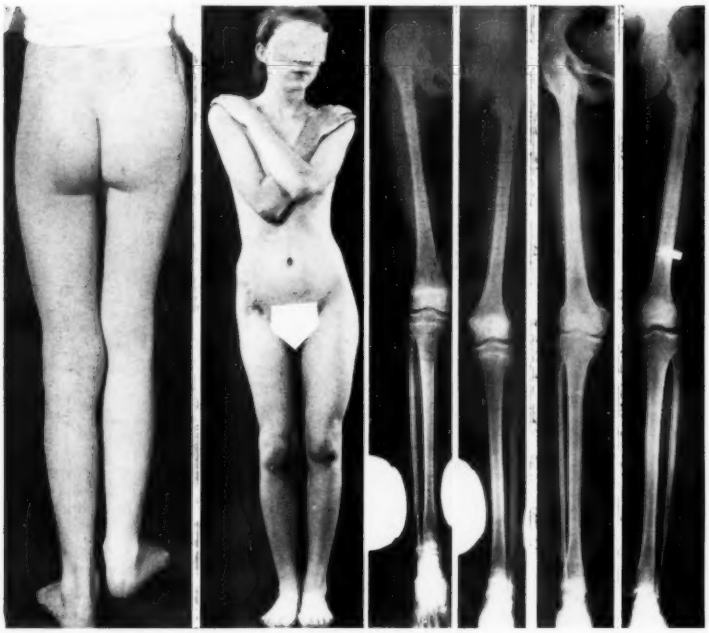

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Fig. 4. Case V. H., illustrative of leg equalization obtained by epiphyseal growth arrest on the distal (left) femur and subtrochanteric osteotomy at the right hip. a, Appearance prior to operation. The right hip is solidly ankylosed due to quiescent tuberculosis; this leg to clinical measurement is 4 inches (10 cm.) shorter than the left. Teleroentgenographic measurements show the right leg to be 3.0 centimeters shorter than the left. b, Anterior view of the same patient, 5 years later. Note that the left knee is slightly higher than the right, but that there is equalization of leg length and a good stance. This result was achieved largely by subtrochanteric osteotomy and abduction of the distal right fragment. c, Teleroentgenograms of both lower extremities taken at the time of the growth equalization procedure. Note that the tibias are of equal length and that the shortening is to some extent due to superior displacement and malposition (adduction) of the left femur. d, Teleroentgenograms of the lower extremities 7 years after growth arrest operation. Note correction of the deformity of the right hip by osteotomy and that the left tibia is now longer than the right. However, in standing position, the level of the ankle joints is approximately the same. Measurement of the teleroentgenograms showed that the patient had actually gained but 1.6 centimeters on the right side.

autogenous bone grafts derived from the excised segment should be added as onlay grafts.

OPERATIVE LENGTHENING OF THE BONES OF THE LOWER EXTREMITY

These operations should be considered only in light of the indications presented by each patient and with full knowledge of the risks involved in operation, as the experiences recorded during the past 15 years have demonstrated the many complications that can arise after these operations have been performed. The detailed technique of operation and the types of apparatus to be used for this purpose will not be described here, as they can be found in the original descriptions by Putti, Abbott (1), Abbott and Crego (2), Moore, Habaush and Finkelstein, Compere (41), Alcorn, West and others (5, 13, 20, 45). The procedures described previously which are more simple are recommended, but there are cases in which the patient, usually already short, is reluctant to sacrifice standing height. The indications for this operation are the following: (1) Leg length discrepancy should be such that it will be totally or almost totally corrected by operative lengthening (between 1 and 3 inches per bone). (2) Adequate

a b c d e

Fig. 5. Another case, J. P., illustrating leg equalization by tibial, femoral, and fibular epiphyseal arrests upon the left leg, but failure to obtain a good functional result due to contractures and malposition at the hips. a, Appearance prior to operation about the left knee. The right leg to clinical measurement is 3 inches (7.5 cm.) shorter than the left. Teleroentgenographic measurements show the femur and tibia on the right to be a total of 4.4 centimeters shorter. b, The same patient 2½ years later. Leg length has been almost equalized at this time, but clinical measurements show a greater discrepancy in functional leg length, due largely to adduction at the right hip, abduction at the left hip, with corresponding pelvic tilt. c, Knee joint roentgenogram—showing appearance immediately after operation. d, Teleroentgenographic prints of the lower extremities prior to operation. Note that the shortening is both in the femur and in the tibia. The right leg is 4.4 centimeters shorter by measurements from the tip of the trochanter to the distal tibial articular cortex. Note also loss of substance of the head and neck of the femur and the adduction deformity at the right hip. e, Teleroentgenograms of the lower extremities taken 3 years after the growth arrest operations. Note elongation in all the bones of the legs. The femurs are of practically equal length as measured from the superior acetabular articulation to the intercondylar notch. The left tibia is now longer than the right. Measurement of the teleroentgenograms show the right leg to be 1.0 centimeter longer, a total gain of 5.4 centimeters, due to the growth arrest operations. This growth occurred in the age span from 13 to 16 years.

musculature should be present about the hip, thigh and calf. (3) Bone must be suitable for the lengthening operation. It should not be done when osteomyelitis has caused the shortening or when the bone is markedly atrophic from any cause.

The operative principles which should be employed are the following: there must be complete control of both fragments by pins inserted through the bone which are mounted in a suitable apparatus to regulate angulation and the rate and extent of lengthening. When the operation is performed, stripping of the periosteum should be limited to the bony area involved in the osteotomy and the length of the osteotomy should not greatly exceed the desired lengthening. An oblique or preferably a Z-osteotomy may be performed. In case of the leg certain soft tissue structures and the fibula should be incised to avoid angulation and other complications. The modification described by Compere (15), consisting of the addition of a large tibial bone graft to the osteotomy site, is an advantage, as this extra bone shortens the period of disability and lowers the incidence of malunion and nonunion. It is illustrated in Figure 3, which shows a femoral lengthening before and after extension. Abbott and Bost (3) recently modified the tibial lengthening technique in light of cadaver experimentation, so that the tendency to angulation of fragments is lessened and a satisfactory outcome is more likely.

RESULTS INCLUDING ILLUSTRATIVE CASES

Epiphyseodesis. This operation has been applied to over 120 patients at the University of Chicago Clinics, many having had more than one

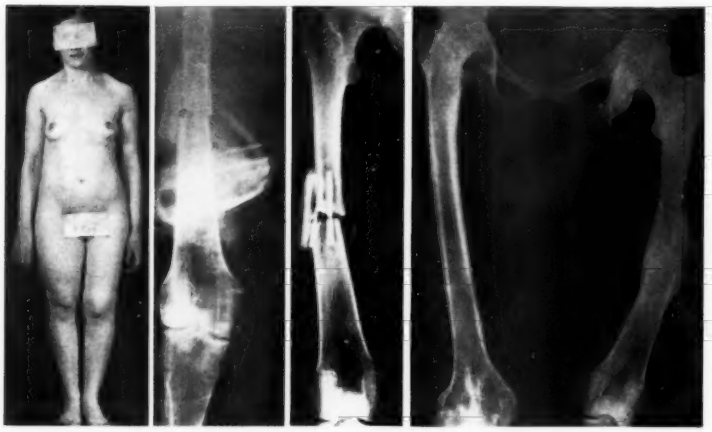

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Fig. 6. Case A. C., illustrating operative shortening of the longer femur. a, Appearance of patient 4 years following shortening of the right femur. Symmetry of the body is good. b, Anteroposterior roentgenograms of the shortened right femur in the cast immediately after operation and on the occasion of a cast change. c, Composite roentgenograms of the femurs 4 years after the femoral shortening.

epiphyseal line closed. There have been no postoperative infections or deaths. The short leg has overgrown the sound leg upon which the operation was performed in 2 patients, one by a half inch and the other by an inch. There was no resultant limp or other disability in these 2 cases, as they were able to compensate by tilting the pelvis. In several cases, especially in girls, the desired retardation of the sound leg was not obtained, as the operation was performed too late. The general tendency has been to operate too late rather than too early in the growth period and to arrest growth at the knee only in the femur when now the tibial and fibular epiphyses would also be attacked.

V. H. (Fig. 4) was 11 years of age at the time, February 10, 1932, of closure of the lower femoral epiphysis of the sound left limb, which was 4 inches (10 cm.) longer to clini-

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Fig. 7. Case R. B., illustrating femoral and tibial lengthening. a, Appearance of patient prior to operation. b, Patient 1 year later after lengthening of tibia and femur. c, Lateral roentgenograms of osteotomized femur. The leg is supported upon a Braun frame. Note pins through distal and proximal fragments. d, Roentgenogram of femur after consolidation of fragments. e, Anteroposterior and lateral roentgenograms of osteotomized tibia and fibula. Note the double sets of pins. f, Anteroposterior and lateral views of tibia and fibula after consolidation of fragments.

cal measurement. The actual discrepancy in the bones was but 3.0 centimeters. This shortening was due to tuberculosis in the contralateral hip, which was firmly ankylosed in some adduction at the time of the operation for growth equalization. The legs were found equal in length on examination January 1, 1935, the patient having been able to discard her built-up shoe 8 months previously. In the interval a subtrochanteric osteotomy had also been performed to correct the adduction deformity. When last seen, April, 1939, her gait was excellent, the tuberculosis process still quiescent, and she was well satisfied with the results of all the corrective operations. Teleroentgenograms of this patient before and 7 years after operation are shown in Figure 4. The result in another patient is shown in Figure 5.

Diaphyseal resection for shortening the longer extremity. At the time of this report, we have observed 25 patients who have had operative shortening of the femur of the sound extremity, all having more than 2 inches discrepancy due to previous disease or atrophy in the opposite leg. The results have been excellent save in one case in which infection followed with loss of bone grafts and with persistence of slight angulation of the femur. Union, however, occurred in all cases. Some angulation of the upper fragment occurred in 3 other cases, but this was corrected by change of cast before bony consolidation was complete.

A. C., female (Fig. 6), who was 20 years at the time, August, 1936, of excision of 2 inches of bone from the right femoral diaphysis, presented an inequality of leg length of 2¾ inches due to tuberculosis of the opposite hip and knee. The hip had been firmly ankylosed for years, but an injury to the left leg had occasioned an exacerbation of symptoms in the quiescent knee that had been held by a fibrous ankylosis. Resection of this knee was performed in May, 1935, with prompt osseous union. The additional shortening occasioned by the latter operation had led to the development of equinus in the foot of the short limb, and the foot became painful due to excessive strain. Figure 6 shows the appearance of the site of operation immediately following excision of bone from the diaphysis. Union promptly occurred. The appearance of the osteotomy site, almost 3 years later, is shown in Figure 6 c. Her gait was improved as a result of the shortening operation, and pain has been absent in the left foot, as the equinus position is no longer necessary in walking.

Leg lengthening operations. Eleven operations have been performed, 4 on the tibia and 7 on the femur, on 10 different patients. Death from cellulitis and septicemia occurred in 1 patient who had moderate amyloid disease that was undetectable by the Congo red test. Moderate infection, followed by massive sequestration of portions of the femoral diaphysis occurred in 2 other patients. In 1 of the latter, a stiff knee also resulted. Most of these cases have previously been reported by Compere (15) and will not be presented individually here. In one instance, tibial lengthening was performed upon the leg that had undergone operative lengthening of the femur.

R. B., male (Fig. 7), case of Dr. E. L. Compere, was 17 years of age when first seen. At this time the left leg was 6 inches (14.5 cm.) shorter than the right, as a result of atrophy incident to 3 years' immobilization of the left hip by a cast for the treatment of tuberculosis. The cast treatment had not resulted in bony ankylosis, but there had been no pain or other symptoms for 6 years, and there remained 15 degrees of passive motion at the left hip. The tuberculosis was considered quiescent. Both the femur and tibia were lengthened, 8½ months elapsing between operations. The femoral lengthening of 2¾ inches was obtained over a period of 3 weeks. Union was present 2½ months after operation. Two and a half inches increase in length of the left tibia was then obtained through the use of the Moore modification of the Abbott apparatus. The patient was last seen 5 years after the first operation was performed. At this last date he had only 0.5 inch discrepancy in his leg length, as determined by clinical mensuration, and he walked with only a slight limp that was due mainly to the abnormality at the left hip.

SUMMARY AND CONCLUSIONS

1. Cases illustrative of three surgical methods that are used to equalize leg length are presented.

2. The indications and special conditions appropriate for each are enumerated.

3. Epiphyseodesis or epiphyseal arrest is the most conservative surgical method. It has a definite field of usefulness, which is limited to the seventh to the twelfth years (fifteen in boys only), depending on the amount of shortening.

4. More study of growth rates under normal and especially pathological conditions is indicated. Under certain conditions compensatory growth probably occurs and the importance of the capital femoral epiphysis is minimal. When growth is more fully understood and cases can thus be better selected for epiphyseodesis, better results will be obtained. There is some uncertainty as to the degree of equalization that will be obtained, and undercorrection instead of overcorrection has been the most frequent undesirable result. Undercorrection can probably be eliminated by more careful selection of patients and earlier operation.

5. The most exact and universally applicable method is operative shortening of the sound extremity. Leg shortening is the most exact and frequently indicated operation in adults and adolescents. The extent of its usefulness and restrictions in younger children has not yet been satisfactorily determined.

6. Leg lengthening is indicated in certain patients who are short of stature but should be performed only by a limited group of surgeons who are experienced in the field.

The authors are indebted to Dr. Dallas B. Phemister for many helpful suggestions in the preparation of the manuscript and his aid in evaluating the material presented. We are indebted also to Dr. C. H. Hatcher for the teleroentgenograms and teleroentgenographic measurements which he made for us.

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DIVERTICULA OF THE URINARY BLADDER

A Clinical Study of 236 Cases

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ALTHOUGH the medical literature for the last two centuries contains descriptions of diverticula of the urinary bladder, it is only recently, as a result of the routine use of the cystoscope and of cystography, that their clinical significance has been appreciated.

The term vesical diverticulum has come to be reserved for a pouch unrelated to any obvious error of development in the bladder wall, the cavity of which is continuous with that of the bladder through a more or less narrowed orifice. Formerly, the term was used to include definite developmental anomalies, such as a patent urachus, accessory ureters, ending blindly, and the hour-glass bladder; now, however, these conditions are no longer classified or designated as diverticula.

Most of the cases described by the older writers as diverticula were discovered at autopsy; in rare instances, however, diagnosis was made by palpation or at operation. Frequently, diverticula were recorded as accessory bladders. Double bladders and even triple bladders have been described.

HISTORICAL

Before presenting the results of this study, which is based on 236 cases, it might be well to review briefly the historical side of the subject.

Diverticula of the bladder were recognized by Albucasis in the eleventh century, Guy de Chauliac in the fourteenth century (quoted by Neult). Blasius in 1677 and Morgagni in 1769 also report diverticula. Other early writers were Brusière, Lambratti, Fannoni and Malgett.

According to Fisher and Pugh, Morgagni (1682-1771) was the first to place diverticula on an anatomicopathological basis. On the other hand, Herbst accredits this achievement to Lorenz Heister (1683–1758). Heister, who was often quoted by Morgagni, denoted all protrusions of the bladder as diverticula and probably originated the term (quoted by Polkey). Morgagni wrote extensively on bladder saccules and cited the following physicians as having discussed sacculi: Ranlin, Riolan, Collot, Heister, Ruysch, Bergert and Dodnaens. Honsted (1743) advanced the idea of urinary retention and hernia of the bladder mucosa. Polkey made a very complete and detailed review of the history of diverticula, to which those who are interested may refer.

The early writers used such terms as double bladder, bladder cells, bladder cysts, saccules, sacs, pouches, false bladder, etc., interchangeably with the term diverticulum, as describing any cavity connected with the bladder by an orifice. In the literature we find "double bladders" described by Rauling, Riolan, Blasius, and Collot which were undoubtedly diverticula. Fischer states that as late as 1863 Michele Scibelli describes a case of congenital double bladder with a much dilated lower end of the right ureter as a "triple bladder."

The first case of vesical diverticulum successfully operated was that of Czerny in 1896. In 1906 Young found 5 reported cases in which the diverticulum had been successfully excised and added 3 of his own. Six years later Leche reported a successful radical removal of a diverticulum and found 14 cases in the literature.

ETIOLOGY

In a discussion of the etiology of diverticula of the bladder it might be well at the outset to call attention to several well recognized clinical facts: (1) diverticula always or nearly always are associated with obstruction at or in front of the bladder neck; (2) they occur with greatest frequency during the period when prostatic obstruction is common; (3) the rare cases seen in children always have some type of bladder neck or urethral obstruction; (4) the lesion is rare in women since bladder neck obstruction is also rare in women; (5) they are always associated with a thickened bladder wall.

These facts justify the assumption of some sort of relationship between bladder neck obstruction, with its increased frequency of urination, difficulty and straining to overcome the obstruction, with increase in the intravesical pressure and thickening of the wall of the bladder, and the presence of the diverticulum.

Since the earliest knowledge of vesical diverticula the question of their etiology has been a

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subject of debate. According to Morgagni, quoted by Fischer, hypertrophic changes of the bladder wall, caused by hypertrophy of the prostate, were the cause of their production. Cruveilhier was responsible for the theory that the pouch was a hernia of the mucosa through the muscular coat. He termed the condition *hernie tunicaire*, on the ground that he considered its distinguishing feature to be the lack of a muscular coat. Later when this author and others including Virchow, Robelin, and Birch-Hirschfeld, found that some diverticula contained muscle fibers, they advocated the theory that diverticula were of two kinds: those of congenital origin having muscular elements, and those of acquired origin without these elements. But today this classification has been practically discarded.

Besides these theories we have writers who strongly advocate the congenital origin of all diverticula and others who believe that they are all acquired.

Rokitansky was of the opinion that diverticula are found only in bladders having hypertrophied walls and that the mucosa is forced between the muscle bundles, the opening being at first slit-like and then rounded. This forcing out of the mucosa, according to Rokitansky, is due to the fact that interstitial changes in the muscle bundles cause uneven resistance which invites sacculation.

Whether or not there is a congenital factor that plays a rôle in the development of a diverticulum is a point that is still under debate. Many authors make the positive statement that in their opinion there is no congenital factor responsible for the production of the diverticulum. On the other hand, some authors have taken a very definite stand on this question and are of the opinion that a congenital factor plays an important rôle. Day and Martin believe that there are two factors present, namely: embryological defects constituting weak spots in the bladder wall and superimposed obstruction. Pugh concludes that there are congenital spots in the bladder wall in all cases, and Harris advances the theory that probably they are developmental defects in the muscular wall. Judd and Scholl state that diverticula are probably due to an embryological defect weakening the musculature, usually at the base, or the result of a defect hiatus in wall of the bladder.

Rose believes that all diverticula are undoubtedly congenital because an unprotected or direct loose fibrous connective tissue pathway must exist through the bladder wall before herniation results.

Hermann attributes diverticula to the inherent weakness in the bladder wall at some point, thus making sacculation prone to occur with increased intravesical pressure.

Lower states that it is not improbable that there is a congenital predisposition to diverticula, and that their development and clinical recognition during adult life are hastened since their dimensions are greatly increased by any of the factors which would bring about increased vesical distention or increased activity of the bladder musculature.

Hinman states that the vesical diverticulum is the result of three factors, namely, the anatomical, the pathological, and the mechanical conditions.

And finally, I would call attention to the extremely interesting observations regarding the etiology made by Watson 18 years ago. As a result of his studies of the vesical cavity in its progressive development from early fetal life until birth he came to the following conclusion:

"The variation and inequality of the intrapelvic and lower abdominal pressure have been noted in a considerable number of fetuses studied. This has caused the bladder at times to become somewhat distorted and the inner walls of the bladder to become extremely irregular, with ridgelike elevations standing out prominently in several areas of the cavity, but more particularly in the zone about the lateral margins of the trigone. These irregularities, augmented by the general processes of growth, may project into the vesical cavity as definite fingerlike invaginations, or the projections may remain as simple elevated ridges which never develop sufficiently to bridge the lumen of the bladder. If the intrapelvic pressure becomes of real significance early in fetal life, the probability of a bridging of the vesical cavity is much more imminent than when it develops during the later months of intra-uterine life. Varying degrees of these irregularities have been demonstrated in the specimens studied, from a simple ridgelike elevation to those which traverse the entire vesical cavity and touch the mucosa of the opposite bladder wall. In certain instances, when the pressure is more lateral they may form bridges along the same side of the bladder from which they originate. When these fingerlike projections traverse the bladder cavity and touch the mucosa of the opposite side, certain regressive changes occur in the approximating mucosa, as indicated in the poor staining qualities of the epithelium and later by a disintegration of the lining cells which allows the mucosa and a portion of the muscularis of two portions of the bladder wall to lie in apposition."

RELATION OF VESICAL DIVERTICULA TO OBSTRUCTION

The presence of obstruction in cases of vesical diverticula and its rôle in their production has been recognized for centuries, although some authors believe that they may develop without obstruction. In view of the fact that modern urological study shows obstruction in practically all cases, it is fair to assume that the cases reported in the older literature as cases without obstruction were probably insufficiently studied,

the obstruction overlooked, or the cases incompletely recorded.

A review of this series of 236 cases showed that complete data on the question of the presence of obstruction were at hand in 230 cases: in 3 of the early cases the records are incomplete and in 3 cases the diverticula were found associated with extensive carcinomas of the bladder. Therefore, in the 230 cases in which data are at hand, obstruction was found in each instance or in 100 per cent.

TABLE I.—TYPE OF OBSTRUCTION

	Cases	Per cent
Benign prostatic hypertrophy	153	66.52
Median bar	34	14.78
Contracted internal urethral orifice	18	7.82
Carcinoma of prostate	17	7.39
Stricture of the urethra	5	2.19
Congenital valves in posterior urethra	3	1.30

AGE

The largest number of cases of vesical diverticula occurs in the age period after fifty, a time of life when obstruction at the bladder neck is common. It must not be forgotten, however, that diverticula do occur before fifty and that the condition may occur in childhood and even in infancy.

In a previous article published in 1934, I reported 6 cases of diverticulum in children, the youngest of which was 12 days old and the oldest was 2½ years. In 3 of them the diverticulum was due to the presence of congenital valves in the posterior urethra, in 2 it was due to contracted internal urethral orifices, and in 1 case the autopsy records failed to mention anything about a causative factor. Since this article was published I have seen 3 more cases of diverticulum of the bladder in children.

A review of this series showed that 87 per cent occurred after the age of 50 years, and that the largest number occurred in the sixth decade, or in 32.2 per cent; 30.6 per cent in the seventh decade, and 6.2 per cent in the eighth decade.

TABLE II.—AGE

	Cases		Cases
12 days to 10 years	9	50 to 60	40
10 to 20	1	60 to 70	76
20 to 30	3	70 to 80	71
30 to 40	4	80 to 90	15
40 to 50	14	Not stated	3

SEX

Diverticula of the bladder are vastly more common in the male than in the female. This statement has been made a good many times by many authors over long periods of time and I wish to subscribe to it. In this series of cases there were 7 females and 229 males.

Among the authors who stress the great preponderance of males over females may be mentioned: Durrieux, Lurz, Legueu and Papin, Cassuto, Hinman, Judd and Scholl, Lower, and many others. Stirling and Rollings found 4 females in a study of 12 cases, an unusual sex proportion.

A brief summary of the 7 cases in females is given in Table III.

Diverticula may be either single or multiple. The incidence in this series was single diverticula, 137 cases; multiple diverticula, 99 cases.

PHYSIOLOGY

Much interest has always centered around the question of whether or not the diverticulum has the power of contraction and hence the ability to empty. Cabot states that it is not clear whether or not the diverticula in their early stages are capable of emptying themselves and are therefore harmless, or whether they are continually the site of residual urine.

Based on their ability to empty, diverticula have been divided into two groups: those that empty and those that do not. The latter have been called "retention diverticula." Retention of urine in a diverticulum sooner or later predisposes to infection and diverticulitis and often to peridiverticulitis with adhesions to important structures which greatly increases the difficulty in their removal. This phenomenon can be easily demonstrated at the time a cystogram is made by withdrawing the contrast media from the bladder by means of a catheter and taking several roentgen-exposures to see not only if the diverticulum is empty after the bladder has been catheterized, but also to demonstrate by repeated roentgenograms just how long it does take the diverticulum to empty if it belongs to the retention type (Fig. 1).

SIZE

Diverticula may vary in size within wide limits containing from 15 cubic centimeters to several liters (Fig. 2). In some instances the diverticulum may be several times the size of the bladder, so that the diverticulum is mistaken for the bladder and the large diverticulum is at first diagnosed as the bladder and the bladder as the diverticulum. Although this combination of a very large diverticulum and a normal or small bladder does not occur frequently, it is of great importance to bear this possibility in mind when reading the cystograms. I have seen several instances of this kind (Fig. 2). Cases have been reported in the litera-

TABLE III.—DIVERTICULA OF THE BLADDER IN WOMEN

Name Age	Symptoms	Duration	Location	No.	Associated lesions	Residual urine	Method of diagnosis	Urine	Treatment	Remarks
Mrs. H. W. NS	Urgency Incontinence Frequency Nocturia Retention	1 yr. 1 yr. 1 yr. 1 yr. 6 mos.	Mesial to and behind right ureteral orifice	1	Contracted internal urethral orifice Bilateral hydronephrosis	47 ozs.	Cystoscope	Albumen+ Pus+++ Bacillus coli Staphylococcus	No treatment Seen in consultation	
Mrs. S. A. 59	Hematuria Frequency Nocturia Pain and difficulty on urination Urgency Pyuria	4 mos. 3 mos. 3 mos. 8 mos. 10 mos. 5 days	On the right, left and posterior walls	M	Contracted internal urethral orifice Hypertrophied interureteric ligament	Not stated	Cystoscope	Albumen+ Pus+++ Bacillus coli	Suprapubic Cystostomy Wedge shaped excision of bladder neck Resection of hypertrophied interureteric ligament	Complete relief of symptoms
Mrs. M. G. 55	Albumin in urine Backache No urinary symptoms	Not stated Several years	On the left lateral wall near the ureteral orifice	1	Bar? at neck of bladder	15 c.cm.	Cystoscope	Pus++ Bacillus coli	None	Discovered duringroutine cystoscopic examination
Mrs. M. S. 46	Frequency Difficulty Urgency Incontinence Nocturia	5 yrs. 5 yrs. 5 yrs. 5 yrs. 5 yrs.	Leftlateral wall above left ureteral orifice	1	Contracted internal urethral orifice	160 c.cm.	Cystoscope Cystogram	Pus++ Bacillus coli Staphylococcus aureus	Electroresection of bladder neck	Last examination on January 3, 1939 showed no residual urine
Mrs. N. VS. 61	Frequency Nocturia Hematuria Pain and burning on urination Imperative urination	4 mos. 4 mos. 2 yrs. 2 yrs.	Left side of bladder	1	Extensive carcinoma of the bladder No visualization of right kidney pelvis	Not done	Intravenous pyelograms and cystogram	Albumen+ Blood++ Pus+++ Staphylococci	Resection and fulguration of bladder tumor	Discovered during routine study to determine cause of hematuria
Mrs. L. F. 82	Hematuria Frequency Nocturia Difficulty Backache Vulvar irritation	6 mos.	On right lateral wall	2	Papillary carcinoma of bladder	Not done	Cystoscope Cystogram	Albumen+ Blood++++ Pus—few Gram positive rods	Fulguration of bladder tumor	Diverticula discovered during cystoscopic examination because of hematuria
Mrs. I. McN. 57	Hematuria Frequency Nocturia Urgency	6 wks. 2 wks. 3 wks. 6 wks.	Right side of bladder	1	Extensive carcinoma of bladder Right hydronephrosis No visualization of left kidney pelvis	Not done	Intravenous pyelograms and cystogram	Albumen ++++ Blood++ Pus+++ Staphylococci	Electroresection and fulguration of bladder tumor	Discovered during routine study to determine cause of hematuria

M = Multiple.

ture in which the diverticulum held a gallon (Green quoted by Targett, and 5.5 liters in Potherat's case). The small cellules so frequently seen with prostatic obstruction are not considered in this paper. A large diverticulum may be present for a great many years without any deleterious effect on the upper urinary tract whereas a small diverticulum in intimate relationship with the ureter may produce a marked hydronephrosis (Fig. 3).

THE ORIFICE

The orifice is generally round. At times it is not recognized during cystoscopic examination, since severe cystitis may be present and the accompanying large flakes of pus may adhere to the wall of the bladder, covering the orifice, or extensive intravesical hypertrophy of the prostate and the presence of calculi may obscure the orifice. During cystoscopic examination changes may be noted in the orifice which give the impression that the orifice has contractile power, that is, that the orifice opens and closes. Some difference of opinion exists as to whether this is due to a true sphincter action or not. Read doubts the presence of a true sphincter. He states that the fibrous ring at the juncture of the sac and the bladder is elastic, and that its contractions are influenced by the bladder musculature surrounding it, but that the ring itself is devoid of muscular powers.

TABLE IV.—CARCINOMA IN BLADDER DIVERTICULUM

Name Sex-Age	Symptoms: Hematuria	Symptoms: Pain	Symptoms: Frequency	Urine: Nocturia	Urine: Albumen	Urine: Blood	Culture	Associated pathology	Histology	Treatment	Result
F.G.K. M—39 yrs.	+	o	o	o	++	+++	Staphylococcus albus	Hydro-ureter, left Hydronephrosis, left Median bar	Epidermoid carcinoma	Nephro-uretero diverticulectomy 2-2-39	Cystoscopic examination 10-10-39 negative. Severe abdominal pain
K.S. M—64 yrs.	+	o	+	o	+	+++	Bacillus coli	Benign prostatic hypertrophy Papillary carcinoma of bladder	Papillary carcinoma	Fulguration of tumor in diverticulum. Electroresection of Prostate 11-2-38	Cystoscopic examination negative 10-24-39
E.A.B. M—63 yrs.	+	+	+	+	+	++++	Bacillus coli Staphylococcus aureus	Benign prostatic hypertrophy Papillary carcinoma of bladder	Papillary carcinoma	2-22-32 Suprapubic cystostomy Diathermy of tumor Implantation of 100 mgm. radium in diverticulum Electroresection of prostate	12-18-39 A recent communication states that the patient is getting along very well and enjoys good health to extent of doing a full day's work each day
C.B.* M—51 yrs.	+	o	+	+	++	++++		Contracted internal urethral orifice	Papillary carcinoma	Diverticulectomy 300 mgm. of radium placed *in situ* from which diverticulum was removed	

*Reported in the Journal of Urology, Vol. 21, p. 381, March 1929.

On the other hand, some authors have described a sphincteric action. Thus Caulk observed in a female infant of 3 months an opening which at times contracted into a crescentic slit. Deming states that the mouths of diverticula may have a peculiar sphincteric action. Buerger reported a case of diverticulum with a contractile sphincteric orifice demonstrated by repeated cystoscopic examination, and Day and Martin reported a case in which perfect sphincteric action of the orifice was observed.

COMPLICATIONS OF VESICAL DIVERTICULA

The common complication of vesical diverticula is infection. Stone and tumor may occur but they are rare. Tuberculosis and leucoplacia within the sac have also been reported, but are very rare.

1. Infection. When they present themselves for examination many patients show the presence of infection. Whether or not the infection is secondary to the bladder infection or vice versa is difficult to say. Infection once started is difficult to treat, especially if the opening is small and the diverticulum does not empty. A diverticulitis is almost universally followed by a peridiverticulitis, of greater or lesser degree, resulting in dense adhesions to the surrounding structures, such as the ureters, prostate, seminal vesicles, or rectum. In certain cases the infection within the diverticulum has been so severe as to cause rupture.

A case of rupture was reported by Thomas. This diverticulum was also complicated by a carcinoma.

Some authors are of the opinion that the size and location of the orifice is important in subsequent infection or tendency to perforation, and they state that the smaller the orifice and the more dependent in position the sac, the more difficult the drainage becomes, thus making for severity of the inflammatory process. Dense adhesions result and make removal of the sac at times very difficult; this may lead to injuries of the adjacent organs, thus complicating the diverticulectomy.

In my series of 236 cases I have never seen a case in which there was rupture of a diverticulum.

2. Tumors. In discussing tumors in diverticula it is necessary to distinguish between tumors which arise within the sac and those which invade the sac secondarily. Papers reporting benign tumors in vesical diverticula have been published by Buerger, Fenwick, Negro, and Blanc, Perthese, and Targett. A sarcoma complicating a diverticulum was reported by Targett, a sarcomatous papilloma by Leuenberger, and a hemangioma cavernosum by Blum.

Kretschmer and Barber, in a review of the relevant literature in 1929, were able to find 20 cases of primary carcinoma arising within a diverticulum and added 1 of their own. Since this time a number of such cases have been reported in the

TABLE V.—STONE IN BLADDER DIVERTICULA

Name Sex—Age	Frequency	Nocturia	Difficulty	Pain	Hematuria	Duration of symptoms	Residual	No. of diverticula	X-rays	Associated lesions	Urine	Remarks
D. B. D. M—67 yrs.	+	+	0	+	+	10 yrs.	20 c.cm.	‡M.	Stones in right diverticulum§	Benign prostatic hypertrophy Right hydro-ureter Right hydronephrosis Bladder stones	Albumen+ Pus++++ Bacillus coli	§Migration of the stone from the diverticulum into the bladder and from the bladder into the diverticulum
R. R. M—20 yrs.	+	+	0	0	0	5 mos.	200 c.cm.	‡M.	Stone in diverticulum, left	Contracted bladder neck Bilateral hydronephrosis Bilateral hydroureters Left kidney stone Left ureteral stone	Albumen+ Pus++++ Bacillus coli Staphylococcus albus Streptococcus viridans	Migration proved by cystoscopy
F. W. C. M—69 yrs.	+	+	+	+	+	5 mos.	—	‡M.	Stones in diverticula	Benign prostatic hypertrophy Bladder stones	Albumen+ Pus+ Red blood cells+ Bacillus coli Streptococcus hæmolyticus	
F. K. M—67 yrs.	+	+	+	0	0	6 yrs.	100 c.cm.	2	Stone in diverticulum, left	Benign prostatic hypertrophy Urethral strictures Papilloma of bladder	Pus Blood Bacillus coli Staphylococcus epidermis	
I. M. M—74 yrs.	+	+	+	+	+	4 yrs.	2 oz.	‡M.	Stone in diverticulum	Benign prostatic hypertrophy Bladder stones	Albumen++ Pus++++ Blood+ Bacillus coli	
J. C. M—59 yrs.	+	+	0	0	+	1½ yrs.		‡M.	Stones in diverticula	Benign prostatic hypertrophy Cholelithiasis Acute cystitis Arteriosclerotic heart disease Bladder stones	Albumen++ Blood+ Pus+ Bacillus pyocyaneus Staphylococcus	
M. D. M—70 yrs.	+	+	+	+	0	10 yrs.	700 c.cm.	‡M.	Stones in diverticula	Benign prostatic hypertrophy Prostatic calculi Acute prostatitis Acute pyelitis; urethral strictures Bladder stones	Albumen+ Pus+ Blood+ Bacillus pyocyaneus	
A. B.* M— 12 mos.	0	0	+	0	0	2 mos.	—	2	Stones in diverticulum	Contracted internal urethral orifice	Albumen+ Pus+ Blood+	

*Reported previously in Am. J. of Dis. Children, 1934, 48: 842–857.
‡M. = Multiple.

literature, including those of Gill, Briggs, Rathbun, Hicks, Well, Irwin, and Peacock and Corbett.

In 1 of my cases the patient had both a tumor in a diverticulum and a tumor taking its origin on the trigone (Fig. 4).

In this series there were 4 cases in which the diverticulum contained a tumor. They are presented in (Table IV).

3. The presence of calculi in diverticula. The presence of a shadow within the bony pelvis, but outside the bladder area, should at once arouse our suspicion that the shadow may be due to stone in a diverticulum (Figs. 5, 6, and 7).

It is not at all surprising that stones occur in diverticula, since two conditions that predispose to stone formation are present in many of these cases, namely: retention of urine and the presence of a nucleus for the deposit of urinary salts. (Pus, bacteria and desquamated epithelial cells.)

Numerous cases are reported in which one or more stones were found within the sac. The stone may be of the dumbbell variety, partly within the sac and partly within the bladder, the two segments being connected by a narrow neck which is surrounded by the orifice of the diverticulum. Stones of the dumbbell variety have been reported

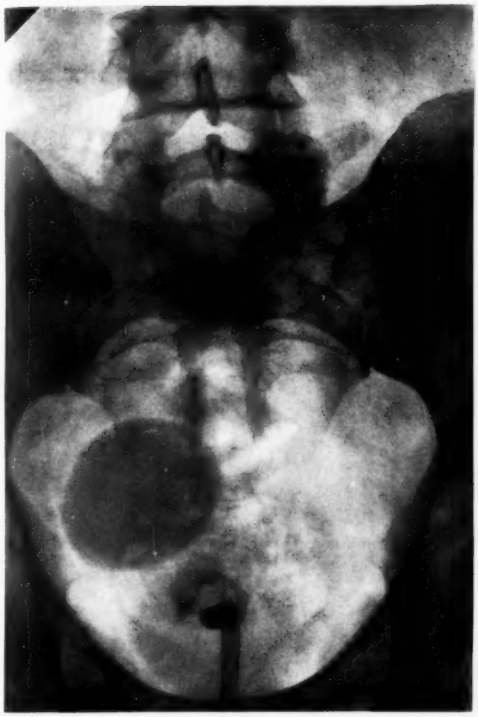

Fig. 1. Retention of media in diverticulum after bladder has been emptied with catheter.

Fig. 2. Showing enormous size of diverticulum which reaches to the fifth lumbar vertebra.

by Davis, Young, Martin, and Crenshaw and Crompton.

Migration of stones from within the diverticulum into the bladder cavity and vice versa is rare, and instances in which this phenomenon has been reported are few. This phenomenon occurred in 1 of my cases (Case 1, in Table V). In this series of cases there were 8 in which stones were found in the diverticula. They are presented in summarized Table V.

4. Tuberculosis. In a few instances tuberculosis occurring in a vesical diverticulum has been reported. As noted by Blum, such cases are due to an extension of the process from a similar infection of the bladder. The latter may be secondary to tuberculosis of the kidney or the genital tract. This condition has been reported by Duvergey, Jeanbrau (quoted by Duvergey), Cornor and Rountree, and Schacht.

Schacht states that tuberculosis is probably the most unusual of all complications of vesical diverticula. His case was revealed at autopsy. The bladder, prostate, and right seminal vesicle were involved in the tuberculous process, as well as the diverticula (three in number). A right nephrectomy for tuberculosis had been done a short while previously.

As far as my personal experience is concerned I have never seen a case of tuberculosis in a bladder diverticulum.

5. Leucoplacia. Leucoplacia has been reported in vesical diverticula. Bugbee reported such a case and stated that a review of the literature revealed 2 other cases of leucoplacia in a diverticulum without involvement of the bladder. Blum, Czerny (cited by Kretschmer in his review of leucoplacia of the bladder) and Stevens also reported a case of leucoplacia in a vesical diverticulum.

In Table VI is given a list of associated lesions found in this series of cases. The incidence of hydronephrosis is not included since many of the patients were seen before the advent of intravenous urography. Likewise the incidence of stones in the upper urinary tract must be incomplete since in the early cases routine roentgen-ray examination was not employed.

SYMPTOMS

There is no symptom or group of symptoms by means of which a positive diagnosis of diverticulum can be made. The clinical picture pre-

Fig. 3. Autopsy specimen. Note compression of left ureter by the diverticulum.

TABLE VI.—ASSOCIATED LESIONS

	Cases
Carcinoma of bladder	14
Bladder stones	13
Stones in diverticulum	9
Kidney and ureter stones	9
Prostatic stones	6
Carcinoma in bladder diverticulum	4
Papilloma bladder	4

sented by the patient with diverticulum is that of bladder neck obstruction. In an occasional case in which the diverticulum is very large a suprapubic tumor can be felt. The presence of a suprapubic swelling, especially to one or the other side of the midline, should arouse suspicion of the presence of a diverticulum. Some authors have called attention to the importance of double voiding as a symptom. The presence of a suprapubic tumor or its gradual diminution in size after the bladder is emptied with a catheter is highly suggestive. Turbid urine or turbid wash water after

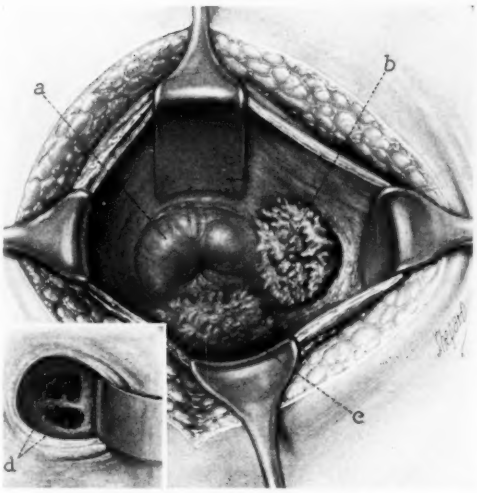

Fig. 4. *a*, Prostate; *b*, tumor protruding from the diverticulum; *c*, tumor on the trigone; *d*, floor of diverticulum after destruction of tumor by fulguration.

the bladder has been emptied with a catheter is also suggestive of a diverticulum that is infected and that is emptying its contents into the bladder. Of special significance is the fact that one is obliged to irrigate for a long time before the wash water returns clear.

It might be interesting to call attention to the fact that the number of cases reported before modern diagnostic procedures were used was negligible, and that since the introduction of modern urological diagnosis the number of cases has greatly increased.

The onset is slow and the symptoms progressive. A point of importance to remember is that the bladder neck obstruction is the factor in causing the symptoms to be prolonged. The duration of the symptoms is given in the following table:

TABLE VII.—DURATION OF SYMPTOMS

Years	Cases
Under one	47
1 to 5	100
5 to 10	49
10 to 15	22
15 to 20	10
Over 20 years	5
Not stated	3

With but few exceptions the patients presented a history characteristic of bladder neck obstruction to which some cases was added the picture of acute or chronic infection.

Fig. 5. Note the large stone in the right diverticulum small stone in diverticulum on left side.

Hematuria may be the symptom that brings the patient to the physician. In some cases it may be the only symptom. Under these circumstances our suspicions should at once be aroused for the possibility of a tumor in a diverticulum (Fig. 8), a tumor of the bladder, or associated pathology in the kidney.

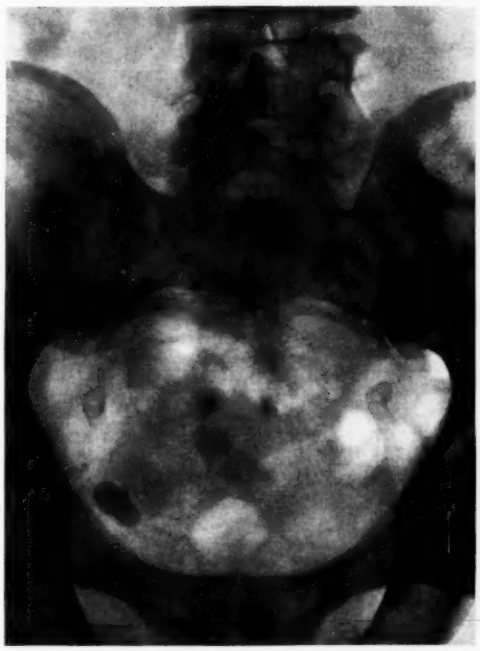

Fig. 6. Stone in bladder. Stone in right diverticulum.

Fig. 7. Moderate sized diverticulum, left. Note collection of media around stone in right diverticulum.

In Table VIII are given the common symptoms and the number of times they were present.

TABLE VIII.—SYMPTOMS

	Cases
Frequency	196
Nocturia	194
Difficulty	141
Lack of force	136
Small stream	132
Hesitation	108
Pain on urination	108
Dribbling	92
Burning	78
Hematuria	78

URINALYSIS

Because of the presence of obstruction, infection is superimposed in the larger number of cases. The association of infection as demonstrated by the presence of pus in the urine was noted in 82.127 per cent of the cases. Pus was present in the urine in 194 cases, no pus was found in the urine in 34 cases, and it was not stated as to whether or not pus was present in 8 cases.

At the present time routine cultures of the urine are made in each case. Unfortunately, in many of the early cases this was not done. No attempt will be made to mention the individual organisms found. In some instances only a single organism was noted and in others a mixed infection was present. In 41 cases the cultures were sterile and in 69 cases no mention of the type of organisms is made.

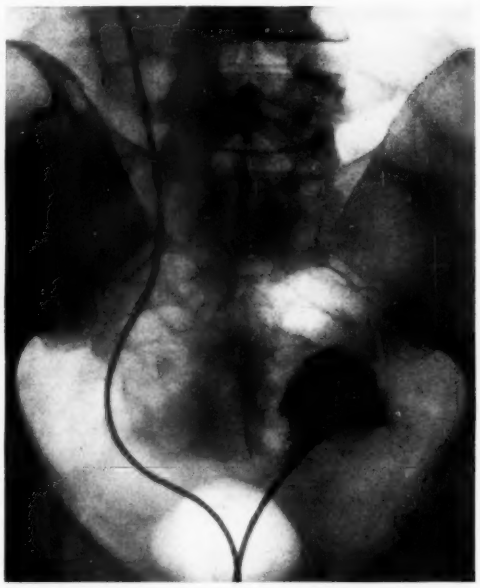

Fig. 8. Note filling defect in lower part of diverticulum due to presence of a tumor.

DIAGNOSIS

Although the various symptoms and signs arising from this disease may serve to direct attention to its presence, or to suggest its possibility, it is only by means of the cystoscope and cystography that a diagnosis can definitely be made. In the larger number of cases cystoscopic examination furnishes an accurate method of diagnosis; however, I think attention should be called to the fact that the opening of the diverticulum may be overlooked in the presence of a very large prostate, severe infection, bladder calculi, and large bladder tumors.

TABLE IX.—BACTERIOLOGY

	Cases
Bacillus coli	79
Eberthella	9
Salmonella	3
Staphylococcus albus	24
Staphylococcus aureus	9
Staphylococcus citreus	1
Streptococcus non-hæmolyticus	23
Streptococcus viridans	2
Streptococcus hæmolyticus	1
Bacillus proteus	9
Gram positive rods	4
Bacillus pyocyaneus	3
Micrococcus flavus	1

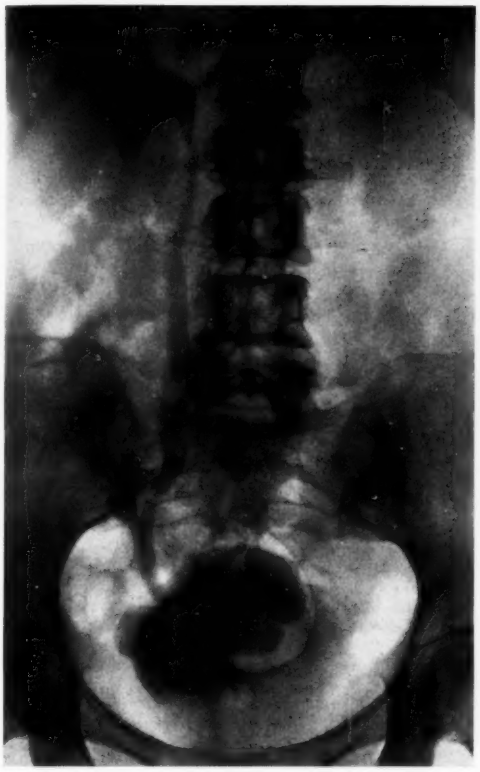

Fig. 9. Showing filling defect in the cystogram due to large carcinoma of the bladder. Left kidney does not visualize. Right side shows hydro-ureter and hydronephrosis. Note diverticulum on right side.

The routine use of cystography will often reveal the presence of one or more diverticula when they are least suspected (Fig. 9.) In an occasional case, the making of a cystogram is followed by chills and fever, and this is probably due to acute pyelitis. This may be due to a reflux of the media used up the ureter and into the kidney pelvis so that a complete urogram is obtained (Fig. 10). The possibility of this phenomenon was emphasized in a previous publication. Of great importance is the fact that not only an anteroposterior film should be made, but a lateral as well, since it is relatively easy to overlook a diverticulum. Contrast cystograms may be made, but I do not use them as routine.

Shadowgraph catheters to outline the size of the diverticulum were formerly used. A catheter in the diverticulum and another one in the ureter may give information of the relationship between the diverticulum and the ureter. After the catheter has been passed into the diverticulum, it may be outlined by injecting contrast media into it.

Fig. 10. Note reflux of media in both ureters. Large diverticulum on the right side. Smaller diverticulum on the left.

In all of our cases of bladder neck obstruction routine urograms were made. As a result of this procedure, diverticula are demonstrated when least suspected. If for any reason a satisfactory outline of the diverticulum is not obtained, a cystogram may be made at a subsequent date. A cystogram is also of value in demonstrating whether a diverticulum has the power of emptying itself, a matter of importance since by this means one can designate between the so called nonretention and retention type of diverticulum. In addition the intravenous urograms show the presence of a unilateral or bilateral hydronephrosis when least suspected.

PROGNOSIS

The prognosis is good provided the sac has been completely excised, the bladder neck obstruction removed, and residual infection eradicated. The vesical diverticulum in itself should not offer a bad prognosis. However, the degree of gravity of the outlook is determined by the amount of associated pathology, which is either the direct or the indirect result of the diverticulum.

The prognosis is influenced by such complicating factors as multiplicity of diverticula,

marked degrees of infection, extensive peridiverticulitis, stones in the sac, carcinomatous changes in the sac, the relation of the orifice of the diverticulum to the ureteral orifices, the relation of the sac to the course of the ureters, renal damage, due to ascending infection and obstruction, and the age and physical condition of the patient.

As is well known, diverticula may be present for many years and not cause damage to the urinary tract; however, this is the exception and not the rule. In isolated instances, large diverticula have been found at autopsy which have caused no bladder symptoms and the patient died as a result of entirely different conditions.

It stands to reason that a large diverticulum must add, except in rare instances, to the gravity of the situation; on the other hand, if a fibrous diverticulum is small and lies in close proximity to the ureteral orifice the damage it causes may be great.

The location of the sac is of great importance. A large sac located in the dome or in the lateral walls does less damage to the kidneys, and, except for the presence of severe peridiverticulitis, is easier to resect.

TREATMENT

The present status of the treatment of diverticula associated with prostatic obstruction is a matter that should invite discussion. Whereas, in the days of suprapubic and perineal prostatectomy, all diverticula were removed, at the present time there is a very definite tendency not to remove them as a routine procedure. As a matter of fact, I no longer perform routine diverticulectomies.

In discussing the treatment of diverticula, it is necessary to assume that the obstruction at the bladder neck has been removed completely. It is well known that, if the obstruction has been completely removed, many diverticula tend to decrease in size and produce no subjective symptoms. The question as to whether the diverticulum or diverticula, if more than one is present, should be removed will depend upon several factors and demands individualization. I would like to discuss this subject under the following headings:

1. Small diverticula. I do not remove small diverticula if the obstruction is removed completely, the urine clear, and subjective symptoms are absent.

2. Large diverticula. Here again, if the obstruction has been completely removed, if the patient is free of symptoms, the urine clear or slightly hazy, due to bacteria and contains a few pus cells, and if the diverticulum is not close to the ureter so as to produce ureteral obstruction, I am of the opinion that by administering urinary antiseptics and keeping the patient under close observation is all that is necessary. However, if the ureter is obstructed, the pyuria does not clear up, and there are recurring attacks of chills and fever, accompanied with urinary symptoms, or if the diverticulum does not empty, then I believe that the patient should have a diverticulectomy.

3. The very large diverticula. In the very large diverticula, one must take into consideration the size and number of the diverticula, whether or not they retain urine and hence are a source of infection, the amount of infection in the urine, and the relation of the ureter to the diverticulum. As a rule, I remove the large diverticula first and then perform the resection.

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THE OBLIQUE MUSCLE-CUTTING INCISION IN ACUTE APPENDICITIS

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WHILE ordinarily the removal of an interval appendix is a simple enough procedure, this is by no means always true of the operation of appendectomy when that hollow muscular viscus is the seat of an acute inflammation. Under these circumstances the satisfactory performance of this operation may tax severely the technical skill of an experienced surgeon let alone that of the occasional operator. In some cases, although the position in which the appendix lies relative to the cecum—always an important consideration in any case of acute appendicitis—has been determined correctly before operation, even to find the appendix may not be easy in spite of the use of the anterior longitudinal band of the cecum as a guide and still further difficulties may await the surgeon when he comes to the actual removal of the offending organ. One of the basic factors essential for the proper performance of any abdominal operation is an adequate exposure of the operative field, this helping the surgeon materially to overcome such difficulties as may present themselves and when an acute inflammatory process is present, such as acute appendicitis, it results in a minimal amount of trauma to the bowel with diminished risk of spreading infection. This short communication is concerned with an incision which affords not only an excellent exposure of the cecal and appendicular regions but also often permits of a virtual extraperitoneal removal of the appendix. Its many other inherent advantages will be mentioned in due course.

This incision is by no means new and while perhaps it is not possible to know exactly who used it first, in the writer's mind it will always be associated with the name of the late Rutherford Morison. At the Royal Victoria Infirmary, Newcastle-on-Tyne, where that surgeon worked and taught for many years, the incision under consideration is always referred to as the Morison oblique muscle-cutting incision. Professor Morison began to draw attention to it in his surgical contributions during the latter years of the last century, about the same time that Charles McBurney first described the muscle-splitting or gridiron incision which bears his name. While the latter incision has rightly attained wide recognition and popularity, for it led the revolt against the median incision in common use at that time by providing a more direct line of attack upon the acutely inflamed appendix, the former, judging from its lack of mention in the literature, appears to be but rarely used and little known. The writer feels that, up to the present, this incision has not gained the recognition it most certainly deserves for it has never been, to his knowledge, either illustrated or described in detail in any of those surgical journals which, by this time, have come to enjoy such wide circulation. The present note is an attempt to remedy that omission and was prompted by a recent series of cases of acute appendicitis which afforded the opportunity of repeated demonstration of the excellence of this incision. The artist's drawings with which the text is augmented are based upon photographs taken during operations and upon the writer's personal sketches.

The incision is begun in the right flank about midway between the costal margin above and the crest of the ilium below and is continued toward the midline parallel to Poupart's ligament for an average distance of 4 or 5 inches. Thus most of the incision lies lateral to an imaginery line drawn between the anterior superior iliac spine and the umbilicus, in contrast to the McBurney incision which lies mostly medial to it (Fig. 1). The external oblique, internal oblique and transversus abdominis muscles, the transversalis fascia and peritoneum are now incised in turn in the direction of the skin incision (Figs. 2 and 3). The only source of moderate hemorrhage likely to be encountered, other than the branches of the superficial epigastric vessels, is the unnamed branch of the deep iliac circumflex artery which is given off near the anterior superior iliac spine and ascends between the internal oblique and transverse abdominal muscles. If looked for, it can be seen easily and hemorrhage prevented by dividing it between forceps. On completion of the wound and retraction of the edges of the peritoneum by the forceps attached to them for this purpose (Fig. 3), certain important features will be noted:

From the Division of Surgery, Sansum Clinic, Santa Barbara. Late honorary assistant surgeon, Royal Victoria Infirmary, Newcastle-on-Tyne.

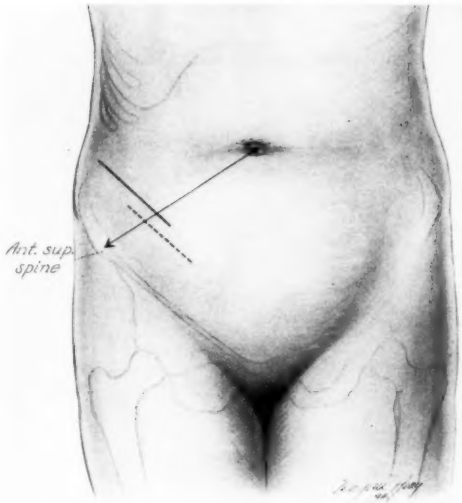

Fig. 1. Approximate and relative positions of the muscle-cutting (shown by continuous line) and McBurney muscle-splitting incisions.

1. A surprising amount of room is provided for surgical manipulations in contrast to that available through the muscle-splitting incision and only occasionally will there be found the need for retractors in the wound. There must be other surgeons who, like the writer, have seen a tense appendix ruptured by the struggle necessary for its delivery through the limited peritoneal aperture which results when the McBurney incision is used.

2. It allows the cecum to be approached from its outer side. Apart from this being of advantage in cases in which the appendix lies in the extracecal position, the advisability of working lateral to the cecum as much as possible in every case is not to be denied since it results in a minimum amount of disturbance of the small bowel with decreased risk of the spreading of infection throughout the peritoneal cavity and of postoperative paralytic ileus.

3. The posterior limit of the peritoneal opening will be found to be near the level of the floor of the paracolic gutter where the lower end of this becomes continuous with the floor or posterior wall of the iliac fossa around and behind the cecum. This feature is of the greatest importance when it is considered necessary or advisable to institute drainage of the peritoneal cavity, for the drain will have its exit as near as possible to the position of maximum dependency for lateral drainage

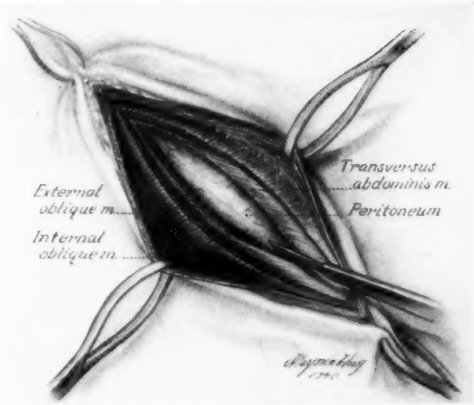

Fig. 2. The incision down to the peritoneum. The external oblique has been incised parallel to its muscular fibers while those of the internal oblique and transversus abdominis muscles have been cut almost at right angles to their course. Note the attachment of the external oblique to the wound towels by catch-forceps; gentle traction on these forceps gives good exposure of the deeper portions of the wound.

of this cavity. There is thus obviated the necessity of making a separate stab-wound in the flank as when a more medial incision, such as the McBurney and especially the paramedian, is used, for to obtain efficient drainage of the peritoneal cavity through either of these incisions is an anatomical impossibility. Nor is the advantage

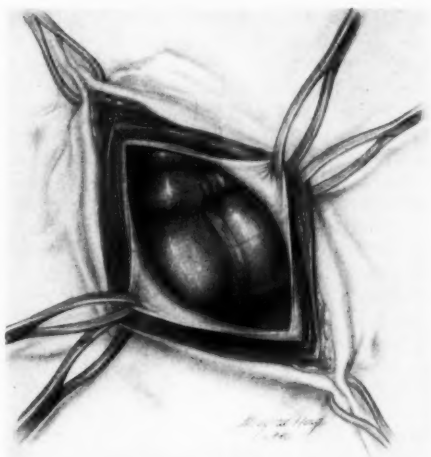

Fig. 3. The muscle-cutting incision completed, and the cut edges of the peritoneum retracted showing the wide exposure of the cecal region obtained.

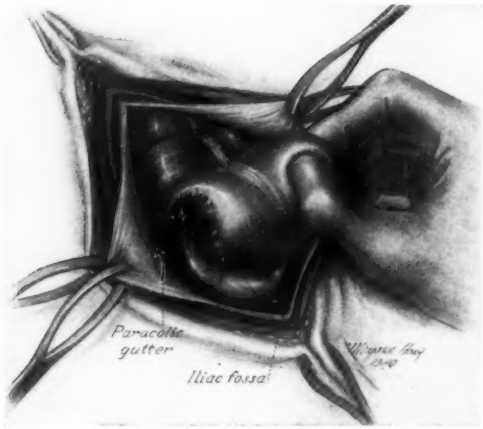

Fig. 4. A typical acutely obstructed and inflamed appendix lying in the extracecal position. Note the exposure obtained simply by slight withdrawal of the cecum. The small intestine is not seen.

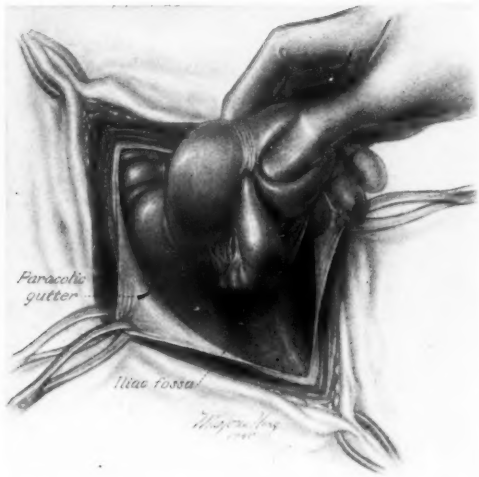

Fig. 5. A retrocecal appendix illustrated to demonstrate the excellent exposure obtained by lifting up the cecum. The small intestine is not seen. This illustration also indicates well the efficacy of the exposure obtained by this incision in cases in which the appendix is of the deeply buried retrocolic type.

of this posterior drainage limited to that of a dependent opening, important as this is, but it allows of a complete closure of the anterior portion of the wound and avoids contact of pus with it, two of the most important preventives of hernia through the cicatrix.

The cecum presents (Fig. 3) and, in cases in which the appendix is extracecal in position, it may be seen and actually removed without even having to withdraw the cecum into the wound (Fig. 4). When the necessity of withdrawing the cecum arises to permit of the best exposure of the appendix, as it does in most cases, this procedure can usually be done with ease and, when combined with gentle traction forward, results in an exceedingly good exposure of an appendix in the retrocecal position (Fig. 5). For the removal of the deeply buried type of retrocolic appendix, it is difficult to conceive of an incision better suited to this purpose; the ample space it provides permits the cecum and ascending colon to be withdrawn forward and upward, thus exposing more and more of the appendix as its distal portion is released. The removal of such an appendix can thus be effected through this incision with an ease impossible of attainment through incisions such as the McBurney or paramedian. When the appendix lies in these situations, or in the subcecal position, it is often possible to remove it without even seeing the small bowel. The removal of the appendix through this incision is therefore often a virtually extraperitoneal operation and this is even more striking when adhesions around the appendix and cecum have already excluded this area from the general peritoneal cavity. Under these circumstances the appendix can usually be removed without disturbing these adhesions and so opening into the peritoneal cavity proper. At this juncture attention is drawn to the advantage of this incision in the treatment of localized appendix abscesses; through it, these can be opened and drained on their outer aspect without fear of breaking down the protective adhesions which are preventing the spread of infection in the peritoneal cavity. The risk of attempting drainage through more medial incisions needs no comment.

When the appendix lies in the so called ileocecal position, pointing upward and to the left behind the terminal ileum, withdrawal of the terminal 2 or 3 inches of ileum together with the cecum increases the exposure (Fig. 6) and is helpful in those cases in which the adhesions holding down the distal end of the appendix are of such density that their separation under direct vision is felt to be advisable. This same maneuver is alone often sufficient to deliver a pelvic appendix into the wound.

The incompletely descended cecum is an abnormality which, while ordinarily only of embryological interest, assumes a surgical importance when the appendix has become the seat of an acute inflammation. In such a case it will be

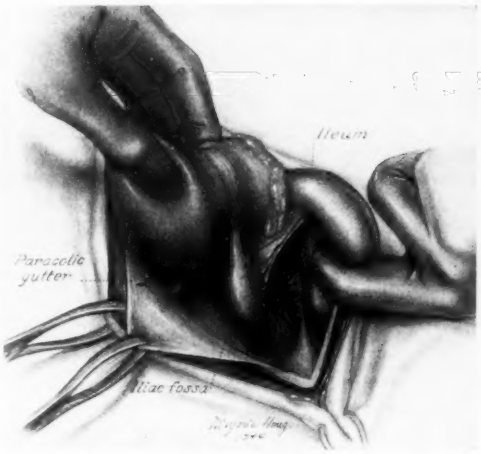

Fig. 6. Appendix in ileocecal position. The cecum is withdrawn laterally, resulting in the terminal 2 or 3 inches of ileum appearing in the wound. The ample room provided by this incision, as indicated here, allows of the delivery of the appendix into the wound with a minimum of trauma and diminishes the difficulties inherent to its removal.

Fig. 7. Gangrenous terminal ileum, cecum, ascending colon, and right half of transverse colon resected with ease through an oblique muscle-cutting incision of average length. The condition of the bowel was due to strangulation by a tight band. This illustration is used to exemplify how a lesion in the right side of the abdomen, other than acute appendicitis, can be dealt with in a satisfactory manner through this incision.

found that the position of the muscle-cutting incision and the ample room provided by it allow the appendix to be dealt with easily when its removal could be accomplished only with the greatest difficulty through an incision of the muscle-splitting or paramedian types.

Because of the fact that acute appendicitis can be closely simulated by other conditions, it is unlikely that there is any surgeon who has not on occasion experienced the chagrin of mistaken diagnosis, for the "acute appendix" may prove to be, not acute appendicitis at all, but some other pathological condition in the right side of the pelvis, the lower ileum, cecum, or ascending colon. Faced with such a situation, the surgeon using the muscle-cutting incision will find in the first place that this incision affords enough room to enable him to make the exploration which is necessary to establish the correct diagnosis—contrast this with the muscle-splitting incision—and secondly, that any such pathology can be dealt with conveniently and satisfactorily through this incision, although sometimes it may be advantageous to extend it medially—an easy matter made easier by remembering to divide the deep epigastric artery between forceps to prevent retraction of its cut ends with resulting troublesome hemorrhage. Ample room is provided for such procedures as removal of a twisted ovarian cyst, ruptured ectopic gestation, or inflamed Meckel's diverticulum, lateral anastomosis between ileum and transverse colon, and even for formal resection of the right half of the colon for carcinoma of the cecum when operation has been undertaken in the belief that the tender mass felt was an appendix abscess. All these procedures have been performed successfully at various times by the writer through this incision, and Figure 7 is an illustration of another, and somewhat unusual, condition recently also dealt with satisfactorily by the same route.

The closure of the muscle-cutting incision is simple and is completed in layers like that of abdominal wounds in general. In clean cases the writer uses continuous sutures for the muscles but when drainage is employed those of the interrupted type are preferred.

One tribunal before which any abdominal incision finds itself is that relating to its liability to subsequent hernia, and in this respect the incision under consideration will undoubtedly be compared at once with the muscle-splitting type. When the abdominal musculature is good and no condition is present or arises to cause increased intra-abdominal pressure of severe degree, it should be axiomatic that any clean abdominal

wound which is sutured carefully and well should never become the seat of a hernia. In regard to the muscle-cutting incision, it has been the experience of the writer first, that this axiom is borne out in clean and uncomplicated cases and second, that should infection of the wound occur, then the chances of development of a hernia are just as great in one type of incision as in the other. There are those who believe that an indirect inguinal hernia may be a late sequel of the muscle-splitting incision because of damage to the ilio-inguinal nerve as it runs its course through the internal oblique at right angles to the line of separation of the fibers of this muscle. In the muscle-cutting incision this muscle is incised parallel to the course of this nerve and injury to it is avoided.

SUMMARY

The oblique muscle-cutting incision is described and illustrated.

The numerous advantages to be gained from the use of this incision in acute appendicitis may be summarized as follows:

1. It affords an excellent exposure of the cecal and appendicular regions and provides ample room for surgical manipulations; these features permit removal of the appendix to be accomplished with minimal trauma to both wound and bowel.

2. The posterior end of the peritoneal opening being near the level of the floor of the paracolic gutter and iliac fossa permits the establishment of a drainage—whenever this procedure is considered necessary or advisable—which is dependent and therefore efficient.

3. In every case the cecum is approached from its outer side; the advantages of this approach are obvious in that: (a) The small bowel is either not interfered with at all or to a minimal degree, thus diminishing the possibility of spreading infection throughout the general peritoneal cavity and of postoperative paralytic ileus. (b) It renders the incision ideal in cases in which the appendix lies in the extracecal or retrocecal or retrocolic positions. (c) When inflammatory adhesions have shut off the appendix and cecum from the general peritoneal cavity, the appendix can be removed without breaking down or disturbing these protective adhesions. (d) The operation is often a virtually extraperitoneal procedure. (e) Localized abscesses can be opened and drained on their outer aspect without fear of spread of infection into the general peritoneal cavity.

4. In the event of symptoms being due to surgical pathology in the right side of the abdomen or pelvis other than acute appendicitis, the incision provides room enough to make the exploration necessary to establish the correct diagnosis and to enable the surgeon to deal satisfactorily with whatever pathology is present.

5. Hernia formation does not follow the use of this incision in clean and uncomplicated cases provided the closure of the wound has been done carefully and well. Should the wound become infected, the incidence of hernia following this incision is no greater than that following other incisions used in acute appendicitis.

CONCLUSION

The many advantages enumerated, to be gained by the use of the oblique muscle-cutting incision whenever operation is undertaken upon the acutely inflamed appendix, have led the writer to hold the opinion that it is the incision of choice in acute appendicitis and they appear to him also to justify the plea he now makes for its more frequent adoption in the operative treatment of this condition.

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THE TREATMENT OF TRIMALLEOLAR FRACTURES OF THE ANKLE

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Minneapolis, Minnesota

SIR ASTLEY COOPER(1) in 1822 first described fractures of the ankle with involvement of the posterior lip of the tibia. In the following century so many excellent descriptions appeared that Ashhurst and Bromer, in their review of the literature on ankle fractures from the time of Pott (1769) to 1922, remarked: "it was with surprise that surgeons . . . saw Cotton (4, 1915) describe this fracture as 'a new type of ankle fracture which has never been adequately described in print and has apparently escaped the notice of those who deal with fractures habitually.'" However, Cotton's description did stimulate interest in this particular fracture and perhaps ultimately led to a more careful consideration of the special problem it presents. Cotton's (4) attention was first directed to trimalleolar fractures—which occasionally but erroneously bear his name—through old malunited cases that came to him for reconstructive surgery. He concluded that unless the fracture with its attendant posterior dislocation is recognized and reduced, "we have to reckon with the meanest and most serious lesion that occurs at the ankle joint." Six years later Cotton (5) published a description of his method of treating the fracture in its fresh state. This consisted of closed reduction by manipulation and application of a plaster cast.

The posterior lip of the tibia was entitled the "posterior malleolus" by Destot in 1911, and Henderson and Stuck in 1935 suggested the term "trimalleolar" for ankle fractures involving the medial and lateral malleoli and the posterior lip of the tibia, or "posterior malleolus." Even though the posterior lip of the tibia, anatomically, is not a malleolus, we believe "trimalleolar" is a convenient descriptive term, and for this reason despite its obvious misapplication, use it to indicate these fractures.

In 1922 Lounsberry and Metz discussed trimalleolar fractures, as well as those with involvement of the anterior tibial lip and were the first to advocate open reduction and internal fixation of the posterior or anterior tibial fragments. They used autogenous bone pegs as the fixative agent.

Since this time, much has been written on the subject and the one notable thing about this considerable literature is the variety of different treatments proposed for trimalleolar fractures. Beck, Deuticke, Key and Conwell, Speed, and others agree that open reduction and internal fixation of the posterior tibial fragment often is necessary when the fragment is *large*. However, just what these authors mean by *large* remains moot, and just how *large* is *large* no one seems to agree. According to illustrative roentgenograms in various reports *large* may vary from less than one-quarter to about three-quarters of the anteroposterior measurement of the inferior tibial articular surface.

Hubmann, Boehler, and Murphy contend that open operation is never indicated and claim uniform success for their manipulative or combined manipulation-traction methods. Boehler and Hubmann use manipulation with three-directional traction by means of adhesive tape or a Kirschner wire through the os calcis with the leg on a Braun frame. Murphy and several others recommend only manipulation followed by the application of various plaster casts.

Dieterle and Felsenreich describe percutaneous nailing by means of single or multiple Kirschner wires to impale the fragment and hold it in position.

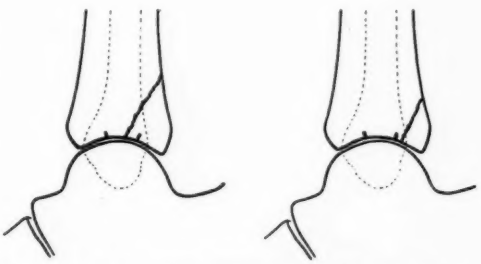
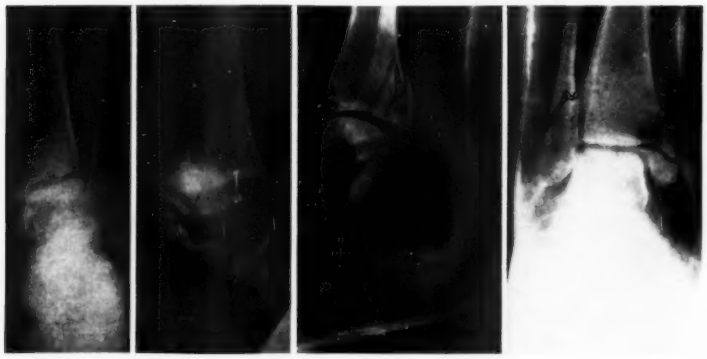


Fig. 1. a, left, The "classical" trimalleolar fracture. The posterior fragment involves *one-third or more* of the *tibial articular surface*. b, The "minimal" trimalleolar fracture. The posterior fragment involves *less than one-third of the tibial articular surface.*

From the Minneapolis General Hospital and the Department of Surgery, University of Minnesota Medical School.



a b

Fig. 2. a, Anteroposterior and lateral roentgenograms of a "minimal" trimalleolar fracture. Note the posterior dislocation of the foot at the ankle joint and the posterior fragment which is less than one-third of the tibial articular surface. b, Roentgenograms of the same fracture which were made after reduction had been carried out. The reduction was obtained by simple manipulation. Although the posterior fragment is not anatomically replaced, the medial and lateral malleoli are, and the patient has a painless ankle, normal motions, and no disability.

MacKinnon adopts a rather hopeless attitude in that he regards fusion of the ankle joint as the procedure most likely to give the best result, while isolated case reports, Storey, offer the use of pillow splints and immediate active motion.

FIVE YEARS OF ANKLE FRACTURES AT MINNEAPOLIS GENERAL HOSPITAL

In the 5 year period ending July 1, 1939, 359 ankle fractures were treated at the Minneapolis General Hospital (Table I). In this series there were 77 "minimal" and only 10 "classical" trimalleolar fractures. These terms were coined by one of us (M. C. N.) only as a convenience in description and to indicate variation in degree of the particular fracture. They are not separate or different fractures but merely different degrees of the same fracture. By "classical" trimalleolar, we mean one in which the posterior fragment involves *one-third or more* of the *articular surface* of the distal end of the tibia (Fig. 1a). Since coining this term, we have found basis for it in the literature in that Tillaux called the posterior fragment in a trimalleolar fracture the "classic" fragment. In contrast to "classical," we use "minimal" to indicate involvement of *less than one-third* of the tibial *articular surface* (Fig. 1b).

Fig. 3a. Antero-posterior and lateral roentgenograms of a "classical" trimalleolar fracture. Note the posterior dislocation of the foot at the ankle joint and the posterior fragment which is greater than one-third of the tibial articular surface.

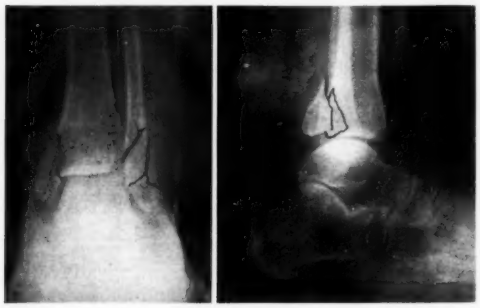


Fig. 3b. Roentgenograms of the same fracture after attempted reduction by all accepted methods except open operation. The patient has only one-third normal motion in the ankle joint and is permanently partially disabled. Moreover the ankle is painful.

TABLE I.—ANALYSIS OF 359 ANKLE FRACTURES

Type of Fractures	Number	Percentage of Total
Lateral malleolus alone	141	39.2
Lateral malleolus with posterior tibial lip	33	9.2
Posterior lip of tibia alone	14	4.0
Bimalleolar	50	14.0
Medial malleolus with posterior tibial lip	10	2.8
Medial malleolus alone	24	6.6
Trimalleolar—Classical	10	2.8
Minimal	77	21.4

MINIMAL TRIMALLEOLAR FRACTURE

These 87 trimalleolar fractures were treated by several surgeons using various standard methods with uniformly good results in the "minimal" cases. Simple manipulation with the knee flexed to a right angle, and application of a plaster cast from the toes to the knee, in the great majority of cases accomplished and maintained reduction. Follow-up study on many of these "minimal" cases disclosed that complete replacement of the "posterior malleolus" is not necessary for an excellent clinical result if the medial and lateral malleoli are accurately reduced. This no doubt is due to the greater width of the anterior portion of the talus, thus preventing posterior luxation of the foot (Figs. 2a and b). The larger the posterior fragment, the more accurate the reduction must be, and in "classical" cases, the reduction must be anatomically perfect before one can expect a good result.

CLASSICAL TRIMALLEOLAR FRACTURE

The 10 cases of "classical" trimalleolar fracture were studied very carefully, and we were able to do follow-up work on 8 of them. Mrs. C. H. was the first case of "classical" trimalleolar fracture treated by the authors at the Minneapolis General Hospital. Manipulations, casts—short, long, with foot in plantar flexion and in dorsiflexion, with knee extended and with knee flexed—three-directional traction according to Boehler, and manipulation with the extremity in traction failed to obtain reduction. The end-result was painful and disabling for our patient (Figs. 3a and b).

This experience stimulated us to search for a better means of handling these difficult cases. Accordingly, when manipulation and plaster cast obtained but failed to maintain reduction in the

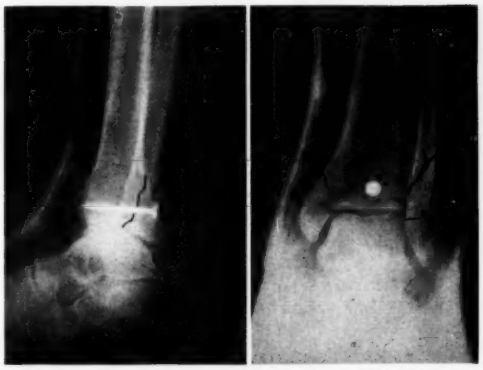
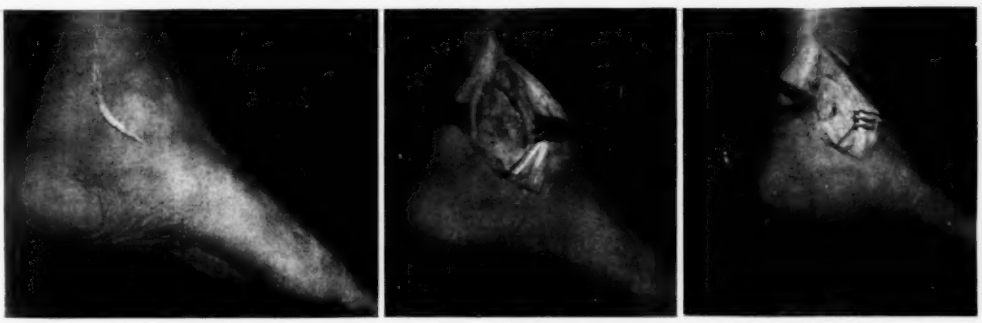


Fig. 4. Roentgenograms of another "classical" trimalleolar fracture after open reduction and internal fixation with one vitallium screw. The patient has normal motions at the ankle and subastragalar joints, no pain and no disability.



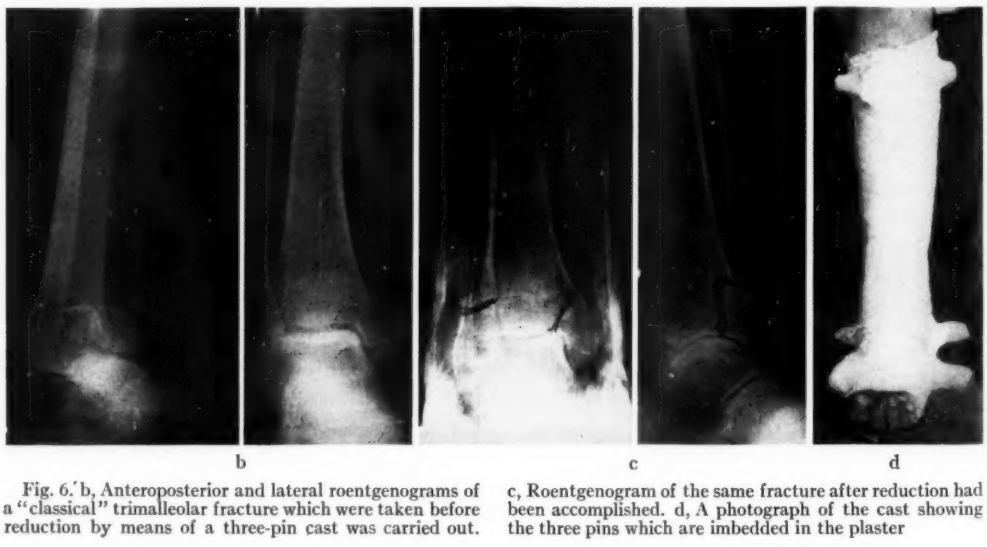
a b c

Fig. 5. a, Photograph showing the incision which is used in the open operation for "classical" trimalleolar fracture. b, The dissection is carried deeper in such a manner as to show the retraction of the vessels, nerve, and tendons and to give exposure of the posterior fragment of the fracture. c, The posterior fragment is shown as it is being fixed in place by vitallium screws after the cortex of the fragment has first been drilled. It is necessary that the screws extend through the entire width of the anteroposterior diameter of the tibia.

Fig. 6a. The bones of the leg in traction apparatus to illustrate our method of obtaining three directional traction. The wire through the tibial tubercle affords traction upward in line with the long axis of the tibia; the wire through the os calcis affords traction downward in line with the tibia; and the wire through the metatarsals affords traction forward in line with the long axis of the foot.

case of Mrs. A. M., we did an open reduction and fixed the posterior tibial fragment in place by means of a vitallium screw with excellent results (Fig. 4). In doing this operation, we use a curved incision with the concavity forward, similar to that of Speed. The incision begins 8 to 10 centimeters above the tip of the medial malleolus and just medial to the tendo Achillis posteriorly, and extends downward to a point level with the tip of the medial malleolus and then curves forward below the medial malleolus. The incision is carried through the subcutaneous tissues by sharp dissection, the posterior tibial vessels and nerve and the tendo Achillis retracted posteriorly and laterally, and the tendons of the flexor hallucis longus, the flexor digitorum longus and the tibialis posterior medially and anteriorly, if necessary, thus exposing the fracture. The posterior fragment is easily and anatomically replaced and securely held by means of one or more vitallium screws. The screw must extend through the entire width of the bone. Preliminary drilling of the posterior tibial fragment after it is reduced makes its fixation very easy. The skin is closed with interrupted silk sutures and a dry dressing is applied. The ankle is then manipulated to obtain reduction of the fractured lateral and medial malleoli and a cast is applied exactly as one does in treating a "minimal" trimalleolar fracture (Figs. 5a, b, and c).

Mrs. M. S. fortunately obtained a similar happy result through a "three-pin" cast in which three directional traction was exerted by means of Kirschner wires through the tibial tubercle, the os calcis, and the heads of the metatarsals to obtain reduction, and the reduction maintained by imbedding these wires in a plaster cast ex-



b c d

Fig. 6. b, Anteroposterior and lateral roentgenograms of a "classical" trimalleolar fracture which were taken before reduction by means of a three-pin cast was carried out. c, Roentgenogram of the same fracture after reduction had been accomplished. d, A photograph of the cast showing the three pins which are imbedded in the plaster

tending from the toes to the knee (Figs. 6 a, b, c, and d).

A two-pin mechanical distraction apparatus was used in this procedure, which is difficult and requires much assistance. In the fourth and last patient treated by us, manipulation as well as the three-pin cast were unsuccessful, and open operation was again necessary. At operation we found the posterior ligaments or the joint capsule completely torn loose so that the fragment was practically free. Here then is a case—proved—in which open operation is unavoidable. The remaining cases were subjected to various treatments by surgeons of long experience, with unfavorable outcome. We believe that the fault here lies with the methods and not with the operators.

It is most significant in this problem that while over one-third of all ankle fractures have involvement of the posterior lip of the tibia, 93 per cent of these fractures can be successfully treated by exactly the same manipulative methods universally successful in bimalleolar fractures. Only 7 per cent of these fractures offer any real problem. These are the trimalleolar fractures with the posterior one-third or more of the distal tibial articular surface split away. Failure to recognize that this fracture demands a different therapeutic approach from a trimalleolar with a large fragment but less than one-third will lead to disaster. In not a single instance did we fail to get good reduction by manipulation in the "minimal" trimalleolar fractures; conversely, not one "classical" trimalleolar fracture was successfully treated by simple manipulation and immobilization. Hence it is imperative in every trimalleolar fracture with a large fragment posteriorly to ask: Is it a "minimal" or a "classical" trimalleolar? Ofttimes this involves measuring the fragment with a millimeter rule. Then, if the posterior fragment is one-third or more of the tibial articular surface, simple manipulation and application of a cast is worse than useless because of trauma incident to the procedure which may prohibit necessary open operation later. These cases must be treated either by immediate open operation or three-directional traction which can be obtained in several ways: By a cast which is appropriately "windowed" to allow application of pressure to hold the foot forward ("push" instead of "pull"); by skin or skeletal traction with the leg on a Braun frame; or by a three-pin cast.

CONCLUSIONS AS TO TREATMENT

1. "Minimal" trimalleolar fracture is a trimalleolar in which the posterior tibial fragment comprises less than one-third of the anteroposterior measurement of the distal articular surface of the tibia. It can in every case be adequately reduced by manipulation under anesthesia with the knee flexed to 90 degrees, and reduction maintained by a plaster cast from the toes to the knee with the foot flexed to a right angle and in mid-position as regards inversion and eversion.

2. "Classical" trimalleolar fracture is a trimalleolar in which the posterior tibial fragment comprises one-third or more of the anteroposterior measurement of the distal articular surface of the tibia. It can be reduced by manipulation, but the reduction cannot be maintained in every case, even if the cast extends from the toes to the groin with the knee held in 90 degrees' flexion. Therefore, it should be opened, preferably within the first few hours when the skin is still in good condition and swelling absent or minimal. We use Speed's incision—just to the medial margin of the tendo Achillis and vitallium screws to fix the fragment in place. A light dressing is applied, the ankle manipulated to reduce the fractures of the medial and lateral malleoli, and the leg is placed in plaster exactly as for the minimal fractures.

3. In some cases of "classical" trimalleolar fracture, three directional traction in a plaster cast will obtain and maintain reduction; but, because this is uncertain, we believe that it is better to operate immediately. Repeated manipulations, manual or combined with skeletal traction, traumatize the skin and by so doing very frequently preclude any open surgery.

SUMMARY

Trimalleolar fracture of the ankle was first described by Cooper over a century ago.

"Classical" trimalleolar is the most difficult of ankle fractures to treat and probably also the most poorly treated. Open reduction and internal fixation was first advocated by Lounsberry and Metz in 1922.

"Minimal" trimalleolar is a common ankle fracture and can be reduced by manipulation and the reduction maintained by a plaster cast. Considerable offset of the posterior fragments does not preclude a good result.

"Classical" trimalleolar is an infrequent but very serious ankle fracture; it is best treated by very early open operation followed by manipulation.

Dr. A. A. Zierold, professor of surgery at the University of Minnesota and chief of the surgical service at the Minneapolis General Hospital, showed constant interest and gave helpful suggestions in the conduct of this study. Mrs. Josephine Poehler of the Department of Social Service of the Minneapolis General Hospital, and Miss Linnea Sunberg, assisted greatly in follow-up studies.

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CARCINOMA OF THE BREAST

Report of Four Hundred Eighteen Cases Treated at St. Luke's Hospital from 1922 to 1933

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THIS series of 3 men and 415 women with primary carcinoma of the breast was treated in the Out-Patient Department and Wards of St. Luke's Hospital during the 12 year period from 1922 to 1933, inclusive. Patients with recurrences following treatment elsewhere and private patients have been excluded as unsuitable. Otherwise this is a totally unselected group and includes every patient with carcinoma of the breast who was treated either medically, radiologically, or surgically, during this period (Table I). No patient was turned away, without treatment, as hopeless. This group of patients, therefore, cannot be compared statistically to selected groups of patients such as those treated by surgery, surgery and irradiation, or irradiation alone. It is believed, however, that a study of this type gives a truer picture of the treatment of breast cancer than will the study of a group of selected, and therefore probably more favorable, cases.

DIAGNOSIS

The diagnosis in the 374 patients operated upon has been confirmed in every case by histological study, and permanent slides are available in all but 2 instances. In these 2 latter cases, frozen section diagnoses of carcinoma were made, radical operations were performed, and both patients subsequently died of their disease. They have, therefore, been included as true cases of carcinoma of the breast.

Although histological grading has not been routinely done in these cases, we are of the opinion that the biology of the growth, as perhaps at least partially indicated by its histological appearance, ranks with treatment as the major determining factor in the outcome of each case.

In the 44 apparently hopeless cases in which patients were treated without operation in the wards and in the radiotherapy department, the diagnosis was made from the clinical appearance of the tumor and the clinical course of the disease. All doubtful cases, especially those who outlived the normal expectancy of their disease, have been eliminated.

From Surgical Division B, St. Luke's Hospital.

Clinical diagnosis of node involvement. In 299 cases in which there is a record of examination of the axilla before operation and in which there was also postoperative histological study of the axillary nodes, a correct clinical diagnosis of involvement or uninvolvement was made in 197 cases, or 65.8 per cent. In 23 cases, or 7.7 per cent, they were incorrectly called "involved" and in 79 cases, or 26.5 per cent, incorrectly called "uninvolved." The error, therefore, in the clinical diagnosis of involvement or uninvolvement of the axillary lymph nodes in this series was 34.2 per cent.

DURATION OF THE DISEASE BEFORE OPERATION

A history of the known duration of the disease on the part of the patient is available in 341 cases in which the axillary nodes were also studied histologically. Analysis in these cases according to node involvement and 5 year cures obtained is shown in Table II. With the exception of the 100 per cent axillary node involvement observed in patients who were known to have had their tumors 3 or more years, it cannot be demonstrated from this series that delay in seeking treatment had any effect either upon the percentage of axillary node involvement or upon the 5 year cures obtained. This simply emphasizes the fact that breast carcinomas, when they have reached the size to be accidentally discovered by patients, are in the main already "late" growths. This, however, should not discourage us from further educating both the laity and the medical profession concerning the necessity for still earlier diagnosis and treatment of breast cancer.

That patients are coming somewhat sooner for treatment is demonstrated by the fact that in the first 6 year period, from 1922 to 1927, 63 per cent of 192 patients sought treatment within a year after they had noticed a lump, while in the second 6 year period, from 1928 to 1933, 72 per cent of 149 patients sought treatment within a year.

AGES

The ages of 360 patients have been analyzed to see what effect this had on node involvement and

TABLE I.—SYNOPSIS OF DATA OF ALL PATIENTS TREATED

	Number	Per Cent
Radical mastectomies	351	
Simple or incomplete mastectomies	23	
No operation	44	
Total number of cases	418	
Postoperative hospital deaths	14	3.7[1]
Axillary node involvement	244	65.2[1]
No axillary node involvement	116	31.0[1]
No record of axillary node involvement	58	3.8[1]
Lost to follow-up under five years	43	10.2[2]
Dead under 5 years	232	
Dead in first year	78	
Dead in second year	58	
Dead in third year	42	
Dead in fourth year	35	
Dead in fifth year	19	
Five year survivals	129	30.8[2]
With axillary node involvement	57	23.3[3]
Without axillary node involvement	68	58.6[4]
No record of nodes	4	
Ten year survivals	44	16.6[6]
With axillary node involvement	17	10.9[5]
Without axillary node involvement	27	38.0[7]
Known local recurrences	60	16.0[1]
In patients with involved nodes	52	21.3[3]
In patients without involved nodes	8	6.9[4]
Bilateral mastectomies	16	4.2[1]

[1]Of 374 patients operated upon.
[2]Of all patients.
[3]of 244 patients with involved nodes.
[4]of 116 patients without involved nodes.
[5]Of all patients treated from 1922–1928.
[6]Of 156 patients with involved nodes.
[7]Of 71 patients without involved nodes.

5 year cures and are shown in Table III. From this table it can be seen that of the 12 patients in their twenties, 11, or 91.6 per cent, showed node involvement and that only 2, or 16.2 per cent, survived 5 years after operation. After 30 years of age, age apparently had little effect either on the node involvement or the five-year survival rate.

BIOPSIES

Biopsies in our cases were taken by the surgeon with the patient on the operating table and draped for the radical procedure. Needle or punch biopsies were not used as we are opposed to them on theoretical and practical grounds. Theoretically we think that it is an unsound practice to needle malignant tumors of the breast or metastatic carcinomas in the axillary lymph nodes and wait several days before radical surgery when other more direct means of obtaining material for histological diagnosis are available. The introduction of a needle large enough to secure cells for histological study must produce undesirable trauma and hemorrhage, not only in the growth itself but also along the line of puncture. While we are willing to admit that in specially trained and unusually competent hands, both of the surgeon and of the pathologist, punch and needle biopsies are probably satisfactory, we think that the teaching of this method as a sound practice for routine use should be discouraged. Patients are repeatedly seen who have had tumors needled by variously trained physicians and surgeons without the securing of proper material for histological study and without arriving at a definite diagnosis. Knowing the difficulty one sometimes has in securing adequate tissue at operation from very small growths hidden in a fatty breast, it is hard to see how a needle biopsy from such a breast can be accepted with any confidence. Our contention is that in any case surgery is necessary; in the first instance in which the biopsy is negative, surgery is necessary to demonstrate that tumor has not been missed by the needling and in the second instance in which carcinoma is found, immediate radical surgery is indicated. Therefore it appears that needling may be dangerous and in any case it is an unnecessary and useless procedure.

TABLE II.—RELATION OF KNOWN DURATION OF DISEASE BEFORE OPERATION TO AXILLARY NODE INVOLVEMENT AND 5-YEAR SURVIVALS

Known duration of disease	Number of cases	With axillary involvement	Per cent with axillary involvement	Five-year survivals	Per cent of 5-year survivals
0– 5 months	168	114	67.8	53	31.5
6–11 months	60	36	60.0	26	43.3
12–23 months	54	38	70.3	21	38.8
24–35 months	33	24	72.7	8	24.2
36–47 months	11	7	63.5	4	35.4
48–59 months	4	4	100.0	1	25.0
6 –71 months	6	6	100.0	1	16.6
72 months	5	5	100.0	3	60.0
TOTALS	341	234		117	

OPERABILITY

Radical mastectomies were performed by 20 different surgeons on 351 patients, and simple mastectomies, or otherwise incomplete operations, were performed on 23 patients. This gives a total of 374 patients, or 89.5 per cent of the entire series, who were thought suitable for some form of surgical treatment. This, of course, does not represent the "operability" of carcinoma of the breast as we see it, but simply represents the total number of patients for whom some type of surgery seemed the best therapy.

In 1928, a series of 238 cases of cancer of the breast were studied at St. Luke's Hospital (2)

and in this group only 144, or 60.5 per cent, were considered "operable." However, this so called rate of operability was undoubtedly too low and was too dependent upon the individual standards of the various surgeons. Would it not be wise for all clinics to abandon the "operable" and "inoperable" classification for breast cancers and simply regard all cases, both favorable and unfavorable, as patients with cancer of the breast needing treatment? In sufficiently large clinics the material will be comparable and the various methods of treatment in use can be evaluated more easily.

POSTOPERATIVE DEATHS

There were 14 postoperative hospital deaths, an operative mortality of 3.7 per cent of 374 patients operated upon. Postoperative pneumonia accounted for 3; wound infections, 3; cerebral hemorrhage, 2; shock, 2; and coronary occlusion, chronic nephritis with anuria, delirium tremens, and pulmonary metastases, 1 each. These patients have been included with those who have died of cancer.

AXILLARY NODE INVOLVEMENT

In 374 patients operated upon, the axillary lymph nodes were involved in 65.2 per cent, uninvolved in 31.0 per cent, and were not examined in 3.8 per cent. The latter group is composed mainly of patients on whom simple mastectomies were performed for advanced carcinomas. The 31 per cent of uninvolved nodes is therefore the more significant figure.

IRRADIATION

Pre-operative irradiation has not been used in any case in this series for 2 reasons: (1) because this method of treatment was not being used to any extent during the years 1922 to 1933 when this group of cases was seen and (2) because we are not convinced that it is a desirable method of treating breast carcinoma. Adair, who has had a large experience with pre-operative irradiation, furnishes us with the following list of its practical disadvantages; (1) pulmonary fibrosis; (2) poor wound healing, especially separation of wounds; (3) increased technical difficulty of performing the operation after irradiation; (4) increased incidence of postoperative edema of the arm; and (5) fibrosis of the arm muscles with limitation of use. In order to recommend a method of treatment with these known, undesirable results, there must be some well known and proved advantages to offset them. This, to our knowledge, cannot be found in the literature.

TABLE III.—RELATION OF AGE OF PATIENTS TO AXILLARY NODE INVOLVEMENT AND 5-YEAR SURVIVALS

Age of patients in years	Total number of patients	Number with axillary node involvement	Per cent of axillary node involvement	Five-year survivals	Per cent of 5-year survivals
20 to 29	12	11	91.6	2	16.6
30 to 39	40	28	70.0	15	37.5
40 to 49	132	88	66.6	49	37.1
50 to 59	89	62	69.6	24	26.9
60 to 69	70	46	65.7	31	44.3
70 to 79	16	8	50.0	4	25.0
80 to 89	1	1	100.0	0	0
Totals	360	244		125	34.7

The chief argument which is advanced for pre-operative irradiation is that the operative field can either be sterilized of tumor cells or that the fibrosis around the tumor cells will be so great that the risk of dissemination during the radical mastectomy will be greatly decreased. Concerning the first argument, there is no evidence to show that in even half of the cases the breast can be devitalized of its cancer cells by pre-operative irradiation. In 200 cases reported by Adair, complete disappearance of the tumor was obtained in 47, or 23.5 per cent, and in 104 cases with axillary node involvement, sterilization of the axilla was accomplished in 8, or less than 8 per cent of the cases. We feel that a quicker and by far surer way to sterilize a person of carcinoma of the breast locally is to remove the breast and axillary contents as soon as the diagnosis of malignancy is made. It must always be borne in mind, however, that no method of treatment will be curative once the disease has spread outside the local field.

That the spread from tumors during radical mastectomy following pre-operative irradiation is diminished or completely eliminated, is purely theoretical. Our objection, therefore, to this argument is also purely theoretical. We do not believe that the traumatic spread of cancer of the breast during a radical mastectomy which is carried out in a careful and gentle manner is a factor in the cure of this disease. We have a much firmer conviction that the trauma of every day life, including dressing and bathing, which is daily sustained during a course of pre-operative irradiation and the period of waiting which follows, has much more to do with spread of cancer of the breast than has the manipulation of surgery.

Postoperative irradiation of varying amounts and administered over varying periods of time was given to 205 patients; in 38 of these cases only a few light treatments were given and it is felt that they had no effect, while in the 167 instances remaining larger amounts were given and over longer periods of time. Nine patients began their irradiation after local recurrences were manifest and 196 patients received routine postoperative prophylactic treatment. Thirty-six of the latter group subsequently developed local recurrences. Definite improvement of skin recurrences following irradiation was noted in many instances, no improvement in some, and ulceration without improvement in a few. In one case a squamous cell epithelioma developed on the base of an x-ray dermatitis. There is no case, however, in this series in which a patient with a local recurrence, either in the skin or axilla, has been cured of her disease either by surgical excision of the recurrence, by excision and prophylactic irradiation, or by irradiation alone. All patients, except one, with locally recurrent carcinoma are known to be dead of their disease; the one surviving patient has passed the 5 year period, but has evidence of distant metastases. This does not mean that recurrences should not be treated but merely emphasizes the fact that the curability of cancer of the breast is dependent upon its primary local eradication and that any form of primary treatment is inadequate which does not have complete eradication as its objective.

Postoperative irradiation rarely cures and usually does not prevent local recurrences and therefore does not decrease the mortality from cancer of the breast. It does, however, undoubtedly prolong some lives so that a few patients who would otherwise have died within 5 years after operation have survived several more months to be classed in the 5 year group. In this respect postoperative irradiation may possibly add 5 per cent to the 5 year survival rate of breast cancer. The 10 year survival rate is, however, probably not influenced by postoperative irradiation.

BILATERAL OPERATIONS

Bilateral mastectomies, either simple or radical, were performed upon 16 patients at varying intervals of time, giving an incidence of operable bilateral involvement of 4.2 per cent in the 374 patients operated on. Only 2 of these patients are alive at the present time; one is living with pulmonary metastases, 10 and 4 years respectively after the radical removal of the right and left breasts, while the second is living with local recurrences 10 years after the removal of the first breast and 18 months after the second radical operation. The low rate of curability in this group suggests that in many cases involvement of the second breast is metastatic rather than primary.

FOLLOW-UP

The end-results are known in 375, or 89.8 per cent, of the 418 cases. During the first 6 year period, from 1922 to 1927, 34 patients were lost to the follow-up, while in the second 6 year period, from 1928 to 1933, only 9 patients were lost. While a few untraced patients have undoubtedly survived, we believe that the great majority have succumbed to their disease. This certainly has been the history of so called "lost" patients whom we have recently located. In the great majority of instances it was found that they had either died here in New York City or had returned to homes in other parts of the United States in anticipation of death. Four patients went to Europe within 5 years after operation and have not been traced. So far, then, as the end-result statistics are concerned, we are perfectly content to count the 43 untraced patients as dead of their disease.

RECURRENCES

Locally recurrent carcinoma, either in the skin or axilla, was diagnosed clinically in 60, or 16 per cent, of 374 patients operated upon. In 52 instances these recurrences were in patients with primary axillary node involvement, and in only 8 were they observed in patients without primary axillary node involvement. This demonstrates the effectiveness of radical mastectomy in cases in which the disease has not spread to the axilla and also illustrates the fact that routine postoperative prophylactic irradiation to the axillæ of patients without node involvement is probably unnecessary.

END-RESULTS

Of the 418 patients treated, 14 died in the hospital after operation 232 are known to be dead under 5 years, 43 were lost to follow-up under 5 years, and 129 survived the 5 year period.

Nine patients are known to have died of other conditions and free of tumor less than 5 years after operation and another 11 less than 10 years after operation. However, for statistical purposes, these patients have all been considered as having died of cancer.

The 129 5 year survivals were composed of 128 patients operated upon and 1 patient with a supposedly "inoperable" carcinoma who was treated entirely by x-ray. This patient subsequently died of her disease so there is no doubt as

to the correctness of the diagnosis. The 129 patients, therefore, represent a 5 year survival rate of 30.8 per cent of all patients treated, while the 128 surviving surgical patients represent a 5 year survival rate of 34.2 per cent of 344 patients operated upon for both early and advanced tumors.

Five year survivals were obtained in 23.3 per cent of 244 patients with known axillary lymph node involvement and in 58.6 per cent of 116 patients without axillary node involvement. Forty-four, or 16.6 per cent, of 271 patients treated, and 17.7 per cent of 242 patients operated upon from 1922 to 1928 inclusive, survived 10 years. This group is made up of 38 per cent of 71 patients without node involvement and 10.9 per cent of 156 patients with node involvement.

CONCLUSIONS

1. A series of 418 unselected patients with primary carcinoma of the breast, treated at St. Luke's Hospital from 1922 to 1933, is reported. Included are 44 apparently hopeless cases treated by irradiation alone, and 374 treated by incomplete or radial surgery.

2. An error of 34.2 per cent was made in the clinical pre-operative diagnosis of involvement or uninvolvement of the axillary lymph nodes.

3. Age was not a factor either in the percentage of axillary node involvement or 5 year survival rate after the third decade.

4. Surgical biopsies secured at the time of operation were used in preference to those obtained by the needle or punch methods.

5. The axillary lymph nodes were histologically involved in 65.2 per cent, uninvolved in 31.0 per cent, and not examined in 3.8 per cent of the 374 patients operated upon.

6. Pre-operative irradiation was not used in any case while postoperative irradiation of varying amounts and over varying periods of time was given to 205 patients. Although the latter has been of value in the control of local recurrences and pain associated with skeletal metastases, we believe that it should be given routinely after operation only to those patients with axillary lymph node involvement in whom local recurrences are more likely.

7. The end-results are known in 375 cases, or 89.8 per cent, of the entire group. Fourteen patients died in the hospital following operation, 43 were lost to follow-up under 5 years, 232 are known to be dead under 5 years, and 129 survived the 5 year period. This represents a survival rate of 30.8 per cent of all patients treated and 34.2 per cent of the 374 patients operated upon for both early and advanced tumors. With the exception of one patient living with distant metastases, all others with recurrent tumor in the skin or axilla are known to be dead of cancer in spite of local surgical or roentgen-ray therapy. This emphasizes the fact that the curability of cancer of the breast is dependent upon its primary local eradication and that any form of primary treatment is inadequate which does not have complete eradication as its objective.

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OMPHALOCELE (UMBILICAL EVENTRATION) IN THE NEWLY BORN

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UMBILICAL hernia is a condition which is frequently encountered in childhood. In this abnormality there is a variable-sized defect of the rectus fascia and muscles around the navel, so that a peritoneal sac protrudes forward and is covered with skin. In a somewhat similar but rather rare malformation, which we call an omphalocele, there is likewise a separation of the fascia and muscles about the navel, but in addition there is absence of the skin over the bulging abdominal mass. In the omphalocele, abdominal viscera are displaced forward into the umbilical cord so that the presenting intestines are covered only by peritoneum and amniotic membrane. It is evident that this latter type of abnormality is different from the common umbilical hernia and deserves special consideration because the prognosis and the methods of treatment are quite dissimilar.

Many authors have published reports of cases which they have designated as "congenital umbilical hernia," "funicular hernia of the umbilicus," "massive hernia into the umbilical cord." Indeed, some writers have confused umbilical hernia as it is commonly known with the condition which is the subject of this publication. It is important, therefore, to employ some other decisive term, as "exomphalos," "eventration at the umbilicus," "amniotic hernia," or "omphalocele" for these lesions. This latter name is the one which is routinely employed in our clinic. Notes concerning omphalocele and its treatment have appeared in the literature occasionally, but no one author has encountered more than 4 cases. It seemed desirable, therefore, to present a review and study of the records of 22 such cases which have been seen in the Children's Hospital.

EMBRYOLOGICAL CONSIDERATIONS

During early fetal life the celomic cavity normally has a forward expansion into the base of the umbilical cord. In fetuses 7 to 17 millimeters long, a large part of the intestinal tract is displaced anteriorly into this recess in the umbilical cord where it normally remains until some time between the 32 to 42 millimeter stage. Observations vary concerning the exact time during which the intestines enter and leave this forward extension of the celomic cavity, but for practical purposes it may be said that they normally occupy this position from the sixth to the tenth week of fetal life (Fig. 1). The exact reason for this protrusion is unknown. Most authors agree with the morphological observation of Mall (7) that just prior to the extrusion of the intestines there is marked enlargement of the liver. Therefore, the liver appears to take up so much space in the body cavity that the bowel is forced out into the expansible umbilical portion of the cœlom. Subsequently the lower abdominal cavity grows at an accelerated rate and the intestines are then drawn back within its confines.

The formation of an omphalocele in the newly born child is probably related to an arrest or retardation in development of the abdominal cavity during the third month of fetal life. Thus, if the lower abdomen does not grow fast enough at this period, some abdominal viscera will remain in the base of the umbilical cord, because there is insufficient room for them to return to the abdominal cavity.

Other theories have been advanced as the cause of an omphalocele, but these have had little general acceptance. Ahfield suggested that a persistence of the omphalomesenteric duct would hold the bowel out in the cord and hence the abdominal wall could not normally close at the navel. The presence of the omphalomesenteric duct or its remains (Meckel's diverticulum) has been demonstrated more frequently in these cases than in normal individuals. In support of this theory is our Case 4, in which a Meckel's diverticulum opened on the side of the omphalocele sac.

Aschoff proposed that the condition was best explained by persistence of the fetal concavity of the dorsal spine.

Recently Enbom, on the basis of models of human and opossum embryos, advanced the theory that the intestinal loops are withdrawn into the abdominal cœlom by contracture of longitudinal smooth muscle fibers of the omphalomesenteric artery. Further studies must be made to establish the plausibility of this theory.

From the Surgical Clinic of the Children's Hospital and the Department of Surgery of the Harvard Medical School.

This study was supported by a grant from the Godfrey M. Hyams Trust.

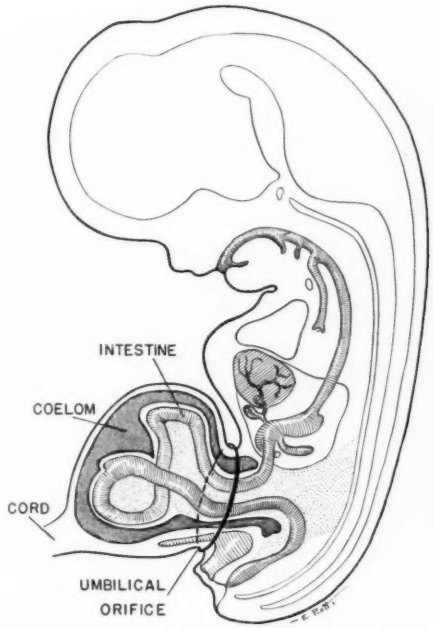

Fig. 1. Sketch from a median sagittal section of a 17 millimeter human embryo, showing the digestive canal (after Mall and Prentiss). There is a protrusion of the midgut out into the expanded base of the umbilical cord. This forward displacement of intestines normally occurs at about the 10 millimeter stage and persists until about the 40 millimeter stage.

After all the evidence presented here is considered, it would appear that the simplest explanation of the etiology of an omphalocele is dependent upon a persistence of the fetal situation wherein there is a disproportion between the size of the peritoneal cavity and its contents. If, after the original protrusion has taken place, the disproportion increases, then more and more viscera are pushed out of the abdomen, particularly those which have not yet attained a retroperitoneal attachment. Probably other factors are occasionally active—as suggested by the numerous other explanations—but these would appear to be of secondary importance.

There is little in the literature to suggest that an omphalocele is a hereditary malformation. None of our 22 patients had any history of a similar condition occurring in their respective families.

CLINICAL FINDINGS

An omphalocele is a thin walled sac at the base of the umbilical cord which may contain intestines, liver, or other abdominal organs. This sac, which varies from several centimeters to as much as 15 centimeters in diameter, has a wide communication with the abdominal cavity. It is a translucent structure composed of peritoneum internally and amniotic membrane externally which appear to be fused when examined grossly. It is usually so thin that it has the transparency of

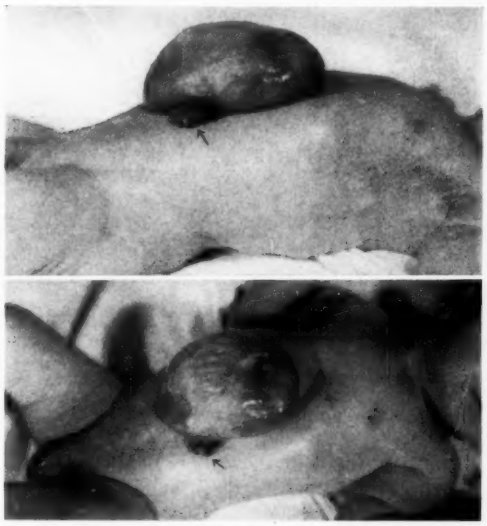

Fig. 2. Case 9. Two day old patient with an omphalocele which contained a portion of the small intestine and a small portion of the right lobe of the liver. Sac completely excised. Patient alive and well. Arrow points to stump of umbilical cord attached to the left side of the omphalocele.

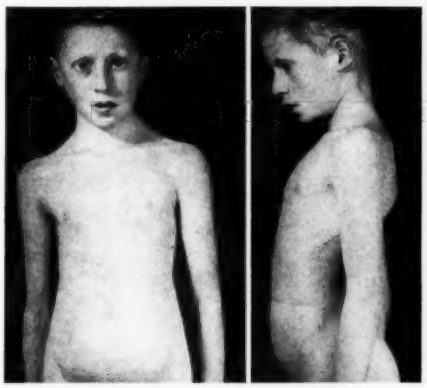

Fig. 3. Case 9. Photograph of patient at 9 years of age showing the condition of the abdominal wall after removal of an omphalocele shown in Figure 2.

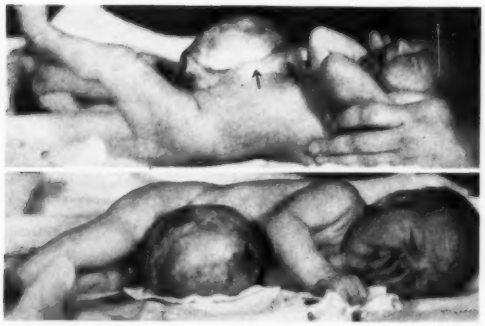

Fig. 4. Case 10. Two day old baby with a large omphalocele containing intestines, large portion of the colon, and a portion of the liver. Arrows point to skin, which is growing over the omphalocele at its base. Remainder of the omphalocele has no cutaneous covering.

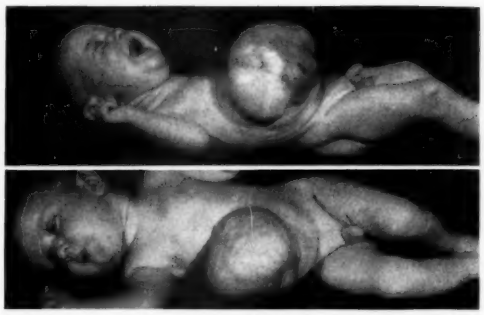

Fig. 5. Case 13. Large omphalocele in a baby 4 months of age. After removal of the sac and replacement of the organs into the abdominal cavity, great respiratory distress and circulatory collapse followed. Child died on the day of operation.

tissue paper and hence the enclosed abdominal viscera can be readily seen through it. The umbilical cord, or its remnant, is attached to the apex of the omphalocele sac, and the two umbilical arteries and the umbilical vein separate and course within the leaves of the sac to enter the abdominal wall as the hypogastric arteries and the umbilical vein. The skin of the abdominal wall usually stops just at the base of the omphalocele, but in some cases it may extend up a centimeter or more onto the sac (Figs. 4 and 11).

The appearance of the sac wall varies with the age of the patient and with the care with which it has been treated by the obstetrician. When seen within the first day of life, the sac is always thin, less than a millimeter in thickness, is quite transparent, very moist and pliable. In such a case its appearance is much the same as the fetal membranes which had encased the baby. When the child is older than one day, or if there has not been a protecting moist dressing, the sac wall tends to be dry, to become less pliable, and to be opaque. This grayish membrane will crack or rupture if carelessly handled. When the child is a few days of age the sac shrivels and a portion of it may become necrotic because of the poor blood supply to its wall. Parts of the wall may then become thickened, blackened, or may rupture. At this stage the sac is often infected, and cellulitis may appear along the surrounding cutaneous margins.

The size of an omphalocele mass can be judged from the accompanying figures and also by reference to the table in which the findings in our 22 cases are listed. The smallest sac which we have encountered was 4 centimeters in diameter, and the largest was 15 centimeters in cross dimension, with an average of about 8 centimeters. In all but 2 patients there was small intestine in the sac. In almost one-half of the series some portion of the liver was outside of the level of the abdominal wall and was protruding into the omphalocele. In 5 cases more viscera were seen in the omphalocele,

Fig. 6. Case 14. Omphalocele in a 3 hour old baby which contained only loops of small intestine. Defect in the abdominal wall was very small. Arrow points to stump of umbilical cord. Surgical treatment followed by successful result.

Fig. 7. Case 14. Photograph of patient 11 days after removal of omphalocele which was shown in Figure 6.

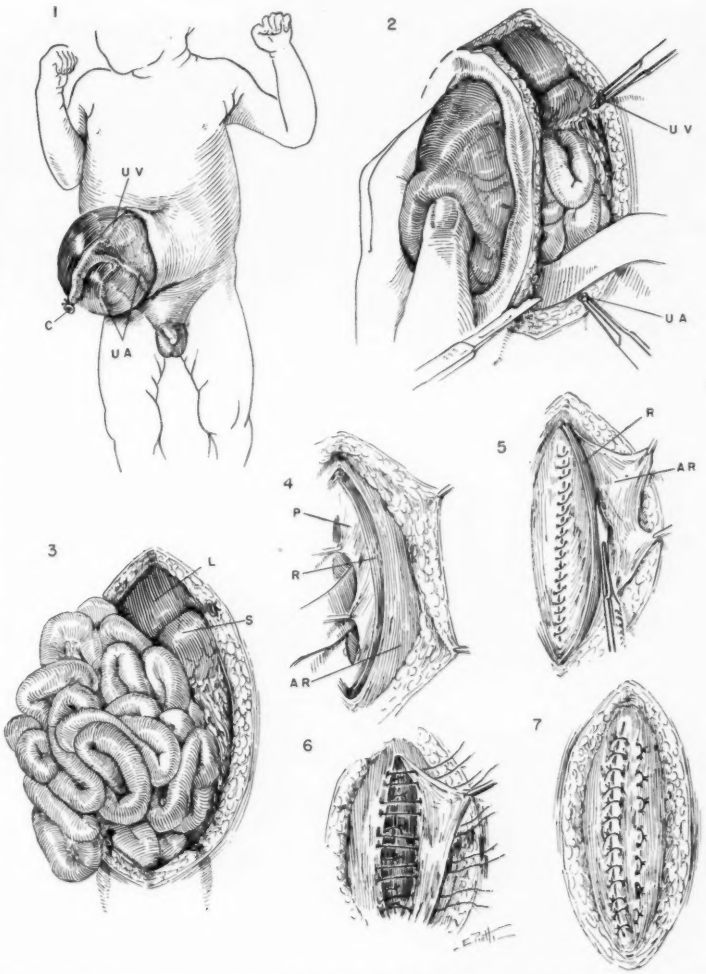

Fig. 8. Sketch of operative removal of an omphalocele. 1, Position of omphalocele on abdominal wall; 2, Method of removal of the sac; cutting away a narrow rim of the adjacent skin and abdominal wall in order to freshen these edges. It is best to cut through the entire abdominal wall at one site and then continue this incision around the sac, cutting through all layers of the abdominal wall with each subsequent sweep of the scalpel or scissors. The large umbilical vein and umbilical arteries must be immediately clamped as shown, in order to avoid hemorrhage. 3, Entire sac has been removed. Umbilical vein and arteries are ligated; intestines prolapse out of the wound. 4, Intestines replaced in the abdomen; peritoneum has been freed from the overlying structures. 5, Peritoneum has been closed with a running suture. The rectus muscle and the anterior rectus fascia are now freed. 6, Rectus muscle has been approximated with interrupted silk sutures. The anterior rectus fascia, which has been freed, is brought together with interrupted mattress sutures of silk. 7, The imbrication of anterior rectus fascia is completed with interrupted silk sutures.

AR, anterior rectus fascia; *C*, cord; *L*, liver; *P*, peritoneum; *R*, rectus muscle; *S*, stomach; *UA*, umbilical artery; *UV*, umbilical vein.

including pancreas, a Meckel's diverticulum, part of the urinary bladder, uterine tube, and spleen.

These infants rarely show any discomfort or altered physiology from having the abdominal viscera thus dislocated and possibly exposed to a lower temperature. There is rarely any respiratory difficulty. In no case have we seen obstruction of the stomach, intestine, or colon before operation. In no instance has there been jaundice or evidence of biliary obstruction, in spite of great dislocation of the liver and obvious distortion of the structures in the gastrohepatic ligament.

Associated congenital anomalies occur with more than coincidental frequency. In one case there was a Meckel's diverticulum which opened on the omphalocele sac. Harelip was present in one child and club-feet were observed in 2 patients. Two had inguinal hernia, one had hydrocephalus, and one had multiple congenital anomalies, including transposition of viscera. One individual had an imperforate anus and two had undescended testicles. Aside from these individuals, all the others appeared to be well developed and to have no other defect.

In 19 cases there was information concerning the time of delivery of these babies. Thirteen of them were born at term, 2 were overterm, and 4 were prematurely delivered.

Fig. 9. Case 15. Illustration of a two day old baby with omphalocele which contained the intestines and a portion of liver. The arrow points to the stump of the umbilical cord.

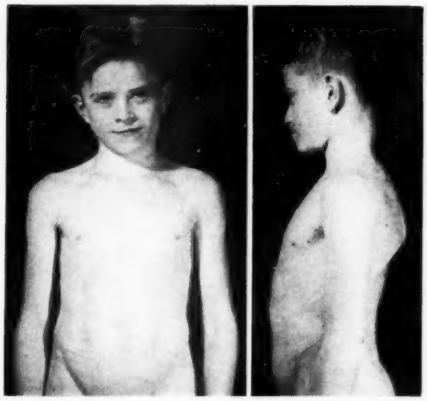

Fig. 10. Case 16. Photograph of patient showing good repair of abdominal wall 9 years after removal of an omphalocele.

TREATMENT

To attempt treatment in these cases by any means other than immediate surgical attack is to expose the child to the imminent risk of a fatal rupture of the omphalocele sac or a spreading infection of the abdominal wall. These babies should have a surgical repair on the first day of life and preferably within a few hours after birth. Food should be withheld from the infant until after operation, for a collapsed stomach and intestine will make the operation easier.

In 1 of our patients, the skin of the abdominal wall had started to grow up into the sac as a narrow phlange around the base of the omphalocele

Fig. 11. Case 18. Four week old child with omphalocele. There is a great deal of skin growing over the lateral and superior portions of the mass, but the central part is ulcerated and infected. Subsequent death from spreading infection.

(Fig. 11). This encouraged us to believe that the epidermis would continue to grow and possibly cover the entire sac. Operation was deferred, therefore, in the hope that such epithelization would be complete, and replacement of intestines into the abdominal cavity and repair of the fascial defects could be done at a later age. Unfortunately, however, infection supervened from which the child subsequently died. There is quite general agreement in the literature that radical operation offers practically the only hope for continued life. Conservative treatment by application of antiseptic dusting powder and alcohol compresses followed by strapping has been reported as being successful on rare occasions, but this should be done only when there is some situation which contra-indicates surgical procedures.

Twenty-two of these patients have entered the Children's Hospital, and 20 of them have been operated upon. The attempt in each instance was to excise the sac, replace the intestines and other viscera into the abdominal cavity, and to effect some repair of the abdominal wall. In the great majority of cases, considerable difficulty was encountered in pushing these viscera back into place, because the abdomen had never developed sufficiently to accommodate them comfortably. The surgeon's main difficulty, therefore, is that of crowding intestines into a cavity which is not big enough for them and also of finding sufficient tissue to close a large opening in the abdominal parietes.

After the sac and a narrow margin of skin have been cut away, a few small vessels must be ligated around the periphery of the wound, but special attention must be paid to the three main vascular

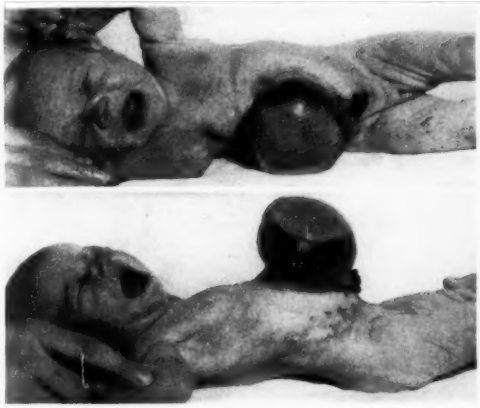

Fig. 12. Case 19. Two day old child with omphalocele which contained intestines and a small portion of the liver.

channels which course from the sac into the abdominal wall. Thus, the hypogastric arteries must be ligated as they enter the inferolateral parts of the abdominal wall. The third vessel, the umbilical vein, usually passes along the left side of the sac and there joins the abdominal wall, where it should be tied.

After the sac is excised, an attempt is made to dissect the various layers of the wall so as to identify the peritoneum, posterior rectus fascia, the rectus muscle, and the anterior rectus fascia on each side of the wound. If sufficient care is taken these tissue planes can all be individually identified and cleared. This dissection of the various layers gives some laxness to the tissues which will be a great aid in repairing the abdominal wall. If enough laxity is present, the edges of the wound can be approximated in layers, which is the ideal procedure. The peritoneum and posterior rectus fascia are brought together with continuous fine chromic catgut of No. 00 or No. 000 size. The bellies of the rectus muscles should be approximated with interrupted sutures, preferably of fine silk. The anterior rectus fasciæ are now approximated in the midline with interrupted silk sutures. It is desirable to imbricate the anterior rectus fascia if possible, but there is usually too much tension to allow such overlapping. If the abdominal wall has been closed without difficulty up to this stage, there is usually no problem in closing the skin, because this is always more lax than are the subjacent layers. In several cases through and through silkworm gut sutures were employed including all layers of the abdominal wall. We have not been convinced that this adds

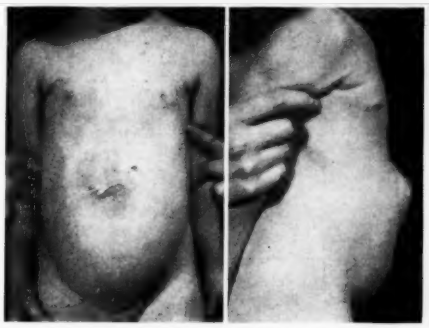

Fig. 13. Case 19. Condition of abdominal wall 2 years after removal of omphalocele shown in Figure 12. There is a muscular defect in the central part of the abdominal wall which will be subsequently repaired at a second operation.

to the strength of the wound or to the improvement of subsequent healing.

In some of the cases closure of the abdominal wall was by no means satisfactory because the great tension and the apparent underdevelopment of the abdominal wall tissues did not permit all layers to be brought together in the midline. In these patients, little more could be done than approximation of the anterior rectus fascia and the skin. In one infant the abdominal wall was so tight that only the subcutaneous fat and skin could be brought together over the defect. This baby has survived and the relatively mild weakness of the abdominal wall (Fig. 13) can be easily repaired when the child is several years of age.

After the crowding of viscera into the abdominal cavity, three serious complications are apt to arise. First, the diaphragm is displaced upward

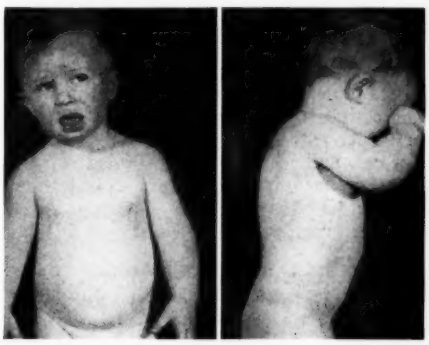

Fig. 14. Case 21. Photograph of patient 2 years after removal of an omphalocele which was 10 centimeters in diameter. The abdominal wall is strong and there is no muscular or fascial defect.

DATA FROM 22 CASES OF OMPHALOCELE OBSERVED AT THE CHILDREN'S HOSPITAL

Case	Age	Sex	Diameter of omphalocele—cm.	Contents of omphalocele				Other congenital defects	Treatment	Result
				Intestine	Colon	Liver	Other viscera			
1	3 days	F	15	+	+	+	Pancreas		Excision	Died
2	3 hr.	F	10	+	o	+			Excision	Died
3	31 hr.	?	?	+	+	o			Excision	Cured
4	4 hr.	M	4	+	o	o	Meckel's diverticulum	Meckel's diverticulum	Excision	Cured*
5	1 hr.	F	5	+	+	o	Bladder and tube		Excision	Died
6	2 days	M	8	+	o	o			Excision	Died
7	6 days	F	8	o	o	+			Excision	Died
8	2 days	F	7	o	o	+		Club feet	Excision	Died
9	2 days	M	5	+	o	+		Inguinal hernia	Excision	Cured
10	2 days	F	15	+	+	+	Spleen		Excision	Died
11	2 hr.	M	8	+	+	o	Bladder	Imperforate anus	Excision	Died
12	2 hr.	M	8	+	o	o			Excision	Cured
13	4 mos.	M	15	+	+	+		Undescended testicle	Excision	Died
14	3 hr.	M	6	+	o	o			Excision	Cured
15	2 days	F	8	+	o	+		Club feet	Excision	Died
16	6 days	M	5	+	o	o		Undescended testicles; hare lip	Excision	Cured
17	36 hr.	F	10	+	+	+			None	Died
18	19 days	M	8	+	o	o		Inguinal hernia	None	Died
19	2 days	F	8	+	o	+			Excision	Cured**
20	5 hr.	M	4	+	+	o		Hydrocephalus	Excision	Cured
21	6 hr.	M	10	+	o	o		Inguinal hernia	Excision	Cured
22	24 hr.	F	5	+	o	o		Transposition of viscera	Excision	Cured

*This case was previously reported by Cutler.
**There is a residual weakness in central portion of abdominal wall which is to be repaired at a second operation.

and respiratory distress and even cyanosis may rapidly appear. Second, great pressure on the inferior vena cava impedes the return of blood from the lower abdomen and legs, so that edema of the legs, circulatory collapse, and even death may supervene. Third, pressure on the stomach and intestines may give rise to partial or temporary obstruction. Operative shock is usually absent or minimal, but the postoperative condition of these small patients is apt to be extremely poor, due to the respiratory distress and the circulatory embarrassment.

PROGNOSIS AND RESULTS OF TREATMENT

If no treatment is given, death will usually result because of rupture of the thin omphalocele membrane. If this accident is not encountered, the delicate sac is apt to become infected and spreading cellulitis of the abdominal wall leads to a generalized infection.

In the present series, 2 patients were not operated upon and both died. Twenty patients were operated upon; 10 of these died and 10 survived. In the 10 living patients, 2 have a weakness in the central portion of abdominal wall which should be readily amenable to later surgical treatment.

The contents of the sac have considerable prognostic importance. The presence of liver within omphalocele is a bad sign. It implies that the abdominal cavity is small, that there will be difficulty in reducing the sac contents, and that there will be considerable crowding of abdominal viscera after operation. Of 8 cases in which some part of the liver was in the sac, there were 6 deaths. If, on the other hand, the sac contains only small intestine, there is usually less difficulty in reducing the omphalocele and the prognosis is accordingly excellent.

The size of an omphalocele has some prognostic value. In those patients with a sac less than 8

centimeters in diameter, there were 75 per cent survivals. When the sac was about 8 centimeters in diameter, there were 28 per cent survivals. And when the sac was larger than 8 centimeters, the survivals were reduced to 17 per cent.

The age of the individual at operation had little to do with the subsequent outcome of the case, excepting that the older individuals were more apt to have infections in the sac and in the operative wound. Adler and Finsterer have both pointed out that the mortality is distinctly lower if operation is performed in the first 24 hours of life, presumably because infection is still minimal in this period. In 3 of our series the sac was infected, and all of these died.

In general, the observations of the surgeon have given considerable information regarding the prognosis. In 9 cases the operator noted that there was a difficult closure and that there was a high intra-abdominal pressure after closure was completed; 8 of these subjects subsequently died. The embarrassment of the respiratory and circulatory systems can well be appreciated by the fact that the 10 post-surgical deaths all occurred within 48 hours after operation. In these fatal cases the postoperative respiratory rates were from 76 to 85 per minute and the heart rates were from 130 to 160 per minute. It is evident, therefore, that little or no attempt should be made to gain an ideal closure of the abdominal wall when intra-abdominal tension is great. In such cases it is probably better judgment to close only the subcutaneous fascia and skin, allow the abdominal wall to stretch, and then repair the fascial and muscular defect 1 week later. We believe that this principle should be employed in future cases.

SUMMARY

The condition of omphalocele or umbilical eventration in the newly born is described. Omphalocele consists of a protrusion of some of the abdominal viscera outward into the umbilical cord so that they are covered with only a thin transparent membrane. Such an omphalocele may vary from 3 or 4 centimeters to as much as 15 centimeters in diameter. The presenting sac is not covered with skin and the thin membrane is apt to rupture and lead to fatal evisceration. The treatment should be by early surgical repair in the first day of life before the omphalocele sac ruptures or becomes infected. The sac and a surrounding edge of skin should be cut away and the organs replaced within the abdomen. Crowding of the abdominal viscera and the deficiency of the abdominal wall usually makes satisfactory closure difficult. If the peritoneum, muscles, and rectus fasciæ cannot be easily closed, it is probably better to suture only the subcutaneous fascia and skin and then secondarily approximate the deeper layers one week later when they will have become stretched.

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THE USE OF A TOURNIQUET IN THE REMOVAL OF TUMORS IN THE POSTERIOR MEDIASTINUM

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IN the removal of solid tumors of the posterior mediastinum such as those of connective tissue or neurogenic origin, it is commonly possible to reflect the parietal pleura from the growth after resection of the posterior portions of several ribs and incision across the intercostal structures. This accomplishes the freeing of the tumor from all but its mediastinal attachments, but since the blood supply is derived from this side, the operator often finds himself in a precarious position as the tumor itself interferes with the exposure and proper control of these vessels.

At this stage of the operation we have found it very convenient to place a braided silk snare about the mediastinal side of the tumor and to draw it tight. By this means, the mediastinal attachment of the tumor is reduced to a pedicle, its vessels are constricted, and the growth itself dislocated somewhat laterally. The tumor can then be removed with perfect hemostasis by incision of its capsule if benign, and enucleation of the mass lateral to the snare. After this maneuver, the operator is left with a dry field in which the vessels to the tumor can be dealt with as necessary, and the serious and sometimes fatal hemorrhage which may result from tearing of the mediastinal structures can be avoided.

The technique is shown in Figure 1. Here, the tumor has been approached through a paravertebral incision with resection of the posterior ends of the third to sixth ribs. The intercostal structures have been reflected laterally and the tumor exposed extrapleurally. The parietal pleura and underlying lung are being retracted and the snare is in place about the mediastinal aspect of the tumor. The insert in the upper right hand corner shows the pedicle after enucleation of the growth and ligation of the vessels and before removal of the snare.

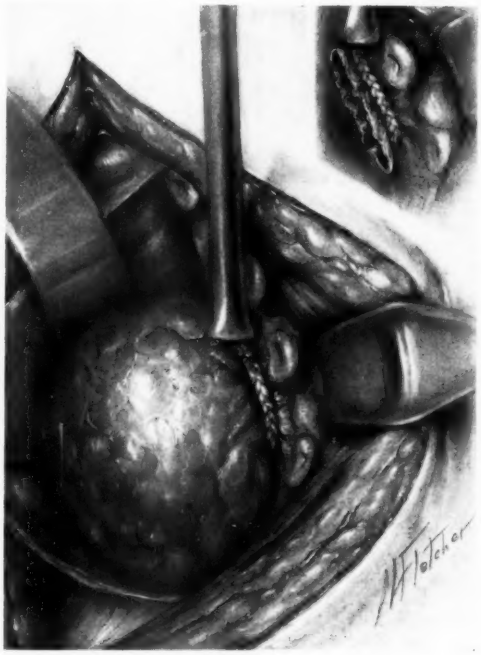

Fig. 1.

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TRENDS IN ANESTHESIA

FOLLOWING the discovery, approximately 100 years ago, of the value of ether and nitrous oxide as anesthetic agents, surgical procedures and methods were developed rapidly. After the original enthusiastic interest in ether and in chloroform had waned and routines for their administration by the open-drop method had been established, the medical profession allowed the practice of anesthesia to lag. Within recent years progress has been accelerated, and the evolution of new agents and methods for their administration has become so rapid that one is justified in saying that a revolution is in process of accomplishment. Several factors have contributed to this trend.

Patients are expecting and demanding more from the medical profession for the relief of pain. Physicians now have a wide choice of agents and methods, and an attempt to meet the demands of patients and of their surgeons is being made. It is no longer necessary for the patient to accept a fixed routine of anesthesia. Agents and methods may be chosen to satisfy demands for adequate sedation and at the same time satisfactory anesthesia may be produced to facilitate the work of the surgeon.

To accomplish this, research has been stimulated and is being actively carried on in the fields of anatomy, pharmacology, physiology, physics, and chemistry. Many new agents have been developed, some have been investigated experimentally and a comparative few have had an adequate clinical trial. Older agents and methods of their administration too are being evaluated statistically in relation to the incidence of postoperative complications. A basis for comparison between the old and the new is being established.

This work has led to the realization that not only is the proper administration of anesthetic agents an art, but that the proper choice of agents and methods should be founded on a scientific basis. To establish criteria for proper selection of anesthetic agents, the collection of clinical and experimental data is continuing. To estimate the extent of interdependence between sets of phenomena, it is essential that negative as well as positive findings be known; a negative finding, not recorded, must be considered unknown. Complete clinical records, therefore, are of paramount importance if the data obtained are to be of any value. In a number of institutions data are being coded in a form suitable for recording on a punch card, to facilitate the sorting and computing of clinical data. As studies progress, opinions regarding certain factors representing cause and effect may be proved untenable, but it is hoped that scientific bases for the proper choice of anesthetic agents and methods of administration will be evolved.

Clinical experience, experimental research, and statistical studies have revealed that certain agents and methods are worthy of inclusion in our accepted list. Methods have improved and their fields of application have been broadened. It is generally agreed that spinal anesthesia has merit for selected procedures, but as yet the agents and methods of application have not been standardized. The technique of carbon dioxide absorption, introduced clinically by Waters, has been widely accepted as a distinct advance in the control of agents administered by inhalation. This development made possible the introduction of cyclopropane to clinical practice. Investigations are being undertaken by a number of groups and methods are being developed to minimize the hazards of inflammability from cyclopropane, ethylene and nitrous oxide with ether added. It is confidently expected that the day is not too far distant when the danger of explosion will be greatly reduced. At the present time intravenous anesthesia can be offered as an alternative for certain procedures; search continues for agents which will produce satisfactory relaxation of the abdominal musculature.

Anesthetists have broadened the field of their endeavor: In the diagnosis and treatment of intractable pain anesthesia plays its part, and in the diagnosis and prognosis of hypertension or of Raynaud's disease, paravertebral injection of procaine is of value; oxygen is administered under controlled conditions for the treatment of pneumonia, abdominal distention, or cardiac decompensation; mixtures of oxygen and helium are administered for the control of asthma. In some institutions recognition of the fact that the anesthetist is usually available at all times has resulted in the creation of an emergency service of which the anesthetist has charge. The administration of blood transfusions and of intravenous and oxygen therapy has fallen within his domain; as a natural sequel in some institutions he has been made responsible for the manufacture of intravenous solutions. He therefore is completely responsible for them, and when untoward reactions occur, as they infrequently do, the cause may be traced and eliminated by him.

In the field of organization, significant progress has been made. The New York Society of Anesthetists was reorganized as the American Society of Anesthetists, Inc. In 1938 the American Board of Anesthesiology, Inc., an affiliate of the American Board of Surgery, was formed and recently, the Advisory Council for Medical Specialties, has recommended that independent status be granted.[1] Following the lead of California, Connecticut, and Indiana, a resolution has been adopted by the house of delegates of the New York State Medical Society granting a section on anesthesia in that organization. This action was followed by that of the American Medical Association in June, 1940, granting a section in that organization.

Anesthetists in their development have gone through the stages of discovery, experimentation, and clinical investigation. Many have developed manual dexterity. Medical regard and public prestige go hand in hand and anesthetists recognize this. Every attempt is being made to warrant the rank of consultant capable of cerebration rather than technician motivated by semiautomatic conditioned reflexes. To accelerate this trend, medical schools throughout the country are urged to include the teaching of anesthesiology in their curricula with courses comparable to those for any other training they provide. Hospitals are gradually demanding higher standards of excellence for members of their staffs practicing this specialty. Every effort is being made among the specialists

[1]Final action not yet taken.

themselves to disseminate knowledge. Centers for postgraduate training have been established. Meetings are being held frequently throughout the country, travel groups have been formed, and finally a new journal *Anesthesiology*, the official publication of the American Society of Anesthetists was launched in July, 1940. Although the specialty is growing rapidly, it is still undermanned. Young physicians who are considering choice of a specialty, would be wise to enter the ranks of the anesthesiologists, provided they are convinced that the work would appeal to them.

RALPH M. TOVELL.

USE AND ABUSE OF TRACTION

THERE is probably no better method of treatment of inflamed joints, no matter what the cause, than traction applied in the right way and in the right amount. Nothing affords relief from pain in this condition, in my experience, so much as properly applied gravity extension. It is useful not only in treatment but in diagnosis. For instance, in a patient with excruciating pain in the region of the hip joint, it is difficult to determine whether the pain is the result of an infection of the hip joint or of an osteomyelitis of the upper end or the neck of the femur. If traction is applied under an opiate by means of a Buck's extension apparatus, and the patient sleeps comfortably without the use of opiates it is evident that an infected joint is present, but if the patient has no relief it is evident that an osteomyelitis is present.

The object of applying traction should be clear to the surgeon; the forceful traction necessary in the treatment of fractured bones and the extension applied for relief of joint pain are aimed at entirely different goals. The amount of traction necessary to reduce or hold a fracture in reduction and the amount of traction necessary to relieve pain in an inflamed joint are entirely different things. In the former condition, our effort is to overcome the pull of muscles which have a tendency to displace the fracture; in the latter, the object should be to relieve muscle spasm caused by irritation of the reflex arc from the joint to the muscles controlling that joint, to take pressure off the inflamed joint surfaces and to put the joint at rest.

The improper use of traction may result in overextension in the wrong direction. Traction should never be used for too long a period or in such a manner that the tissues or nerves are injured. Nerve injury might lead to complete paralysis. In the case of a fracture of the surgical neck of the humerus, for instance, if traction is applied with the arm at a 45 degree angle midway between abduction and adduction, with the elbow slightly forward of the midline or at a level with the anterior plane of the body and the elbow flexed and suspended, the brachial plexus will not be injured. The capsule of the joint probably may be overstretched, but the capsule will usually make some recovery after such overstretching.

In an injury to the cervical spine with fracture but without dislocation, the result of indirect violence to the joints of the neck, it is not unreasonable to assume, that, in view of the construction of the cervical spine, severe traction applied for a long period and pulling on the joint capsule, the ligaments, and muscles of the neck, will damage these structures as much as the original injury had damaged the joints.

Traction is applied to inflamed or damaged joints to relieve the pressure on the joint surface, not to replace any displaced fragments. Overexertion of force on the ligaments and capsule surrounding the joint will produce overstrain and injury in direct proportion to the amount of overweight applied. These

tissues are quite elastic, but no tissue in the body will stand overstrain for a considerable period of time without pain, and a traumatic inflammation can be produced by overtraction treatment as easily as by injury from any other cause.

Traction of the right amount, properly applied, gives more relief in sacro-iliac and lumbosacral arthritis, regardless of the cause, than any other procedure with which I am familiar. Occasionally it takes from 24 to 72 hours to secure complete relaxation of the tissues, but when relaxation takes place the pain is relieved, although it may recur immediately when weight-bearing is resumed unless the inflammation which caused the pain has subsided. If too much weight is applied the ligaments supporting the joints are stretched, the muscles go into spasm instead of being relieved from spasm, and the patient automatically fights the traction. Eight pounds seems to be the amount advisable to be applied to the leg in the form of Buck's extension. For a patient with comparatively weak muscular and ligamentous structure, six pounds is often sufficient. In cervical arthritis, especially of traumatic origin, four pounds is the maximum to be applied.

Traction should always be made in a direct line with the long axis of the bone or bones and joints being treated. If the pulley over which the traction rope runs is too high or too low in its relation to the bed, an angulation occurs. This in itself throws abnormal strain on the joint involved or the joints above or below it. Care should be taken that the right amount of traction is applied and that the pull is at the correct angle. Traction for inflamed joints should give these joints relief; if there is not sufficient pull in the proper line, or if the traction is too great, there will be no relief of pain. The object of this treatment is balance of muscles for the relief of muscle spasm, with a mild separation of joint surfaces without trauma to the supporting capsule and ligaments of the joint.

When traction is used for a fracture, counterbalancing of the pull of the muscles is the objective. The amount of traction frequently must be greater, but it is usually applied in a different way and certainly for a distinctly different purpose. Again it should be stated that the purpose of the traction should be clearly in mind when it is applied.

At all times the traction must be maintained free of any obstruction and in correct alinement. This means at least daily observation. The one responsible for treatment in the surgeon's absence should be cautioned that the weights must not be changed under any circumstances without permission.

A constant smooth pull of gravity may be used for the relief of pain in almost any joint inflammation and, in my opinion, when properly used is one of the most valuable adjuncts in the treatment of joint inflammation and prevention of deformity.

PAUL B. MAGNUSON.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

THE book entitled *Atlas of Surgical Operations* by Cutler and Zollinger[1] is dedicated to and prepared especially for the instruction of the young surgeon in his period of training during his interne and resident years. It is true that textbooks of surgery for the medical student can no longer deal with technique, limited as they must be to a consideration of diseases treated by surgical measures and to the fundamentals of the principles of surgical art and science. It is also true that anyone wishing to learn details of operative technique must consult many works, which often confuse by presenting many methods of attaining the same end. This atlas is designed to present in a single volume the common operations of general surgery and gynecological surgery with sufficient detail so that the technical steps of these procedures will be clear to the neophyte operator.

The book opens with 3 short chapters on surgical technique, anesthesia, and pre-operative and post-operative care. They are philosophical in approach and necessarily, because of their brevity, make no effort to cover these basic considerations with any completeness. The remainder of the work presents operations that have become more or less standardized in modern surgical practice.

True to title, the subject is illustrated by plates, 84 in number, of large size, on which are shown with pen and ink drawings the steps in the operations. The illustrations are by Miss Mildred Codding and in the main she has depicted clearly the minutiæ of the steps in these operative procedures. The illustrations are accurate and usually entirely adequate, but are somewhat colorless, if the term can be applied to black and white, and would gain in attraction and brilliance, if not in usefulness, if the illustrations were made with more contrast. The plates suggest somewhat operations carried out on the cadaver, since in life, tissues abound with contrast and vitality. On the page opposite each plate, instruction is given succinctly without qualification, on the indications, pre-operative preparations, anesthesia, position, operative preparation, incision and exposure, details of operative procedure, closure, and postoperative care, so that easy reference is made from plate to written description.

The authors state that the technique described "emanates from the school of surgery inspired by William Stewart Halsted." The goal is admirable, but Halsted's contributions, great as they were, cannot be demonstrated in an atlas since they consisted of a mode of action and thought rather than an insistence upon anatomical operative details alone. To be consistent in spreading Halsted's gospels in static form, one might well illustrate with Halstedean exactness operative techniques developed by him such as thyroidectomy, radical mastectomy, and herniorrhaphy.

By and large, the operative procedures are described and illustrated so as to be instructive and helpful, and one may state it as a truism that the nearer the surface of the body, the more accurately can the technique be depicted and more precisely carried out by the ready study of diagrams. Some operations, such as the abdominoperineal resection of the rectum, cannot be learned from cuts alone, since drawings and the written word cannot truly give an idea of what the hand feels and does.

The large plates made obligatory a large volume and unfortunately the folio size of the book makes it a mechanical nuisance in any modern library, even though it follow the respectable example of antique surgical literature. It is to be hoped that in the other editions—and it is certain that they will be forthcoming—a more easily handled format will be considered.

Experienced surgeons will undoubtedly differ from the authors on many points and these surgeons will also disagree with one another, but I think all surgeons will agree that the technique shown in the atlas is a safe one and they will feel that the authors are to be congratulated upon striking a usable mean in their selection of methods. One might be critical of illustrating only open types of intestinal anastomoses since there are now in common use several satisfactory methods of making closed union, one of which at least should have been shown to emphasize the importance and availability of the aseptic method. The young surgeon will be on solid ground in following the methods shown until his own experience dictates changes.

Since the work is after all a skeleton, it is to be hoped that it will be used only by those young surgeons whose training gives them succulent tissue of physiology, chemistry, and pathology to cover the dry bones thus presented. For those whose training does not offer this scientific padding, one can only hope that they will realize that pictures do not bleed and that large folio volumes do not die of pneumonia, of embolism, and of shock.

The authors and the illustrator are to be congratulated upon presenting a volume that will be of tremendous value to the young surgeon in the midst of a sound training and to all of these, the book is heartily recommended. It has a place on the table of every hospital library where young men are

[1] ATLAS OF SURGICAL OPERATIONS. By Elliott C. Cutler and Robert Zollinger. New York: The Macmillan Co., 1939.

learning the arts of surgery.

FREDERICK A. COLLER.

THE condensed encyclopedia, *Modern Medical Therapy in General Practice*, edited by Barr[1] will be useful to have at hand in everyday practice. It consists of three volumes of 1,200 pages each, light in weight for their size, and conveniently indexed. The articles are brief and are written by authorities who have firsthand knowledge of the subject and who, usually, have contributed original research to the field they are discussing. In addition to therapy of all medical diseases, that of genito-urinary, gynecological, neurological, ear and eye specialties, is also included. General procedures such as psychotherapy, physiotherapy, and dietotherapy among many others, are thoroughly presented, occupying more than half of volume I. In general, the work can be heartily recommended both for its completeness and for its reliability. PAUL STARR.

THERE is a growing impression among the leaders of the medical profession that malignant disease is not given the consideration it deserves. Those who see many cases in both clinic and private practice observe far too many instances of careless or ill advised medical attention. The American Society for the Control of Cancer is successfully promoting a program of lay education that has a far reaching effect. However, much of its benefits are nullified by a lack of the proper conception of malignant disease by a large percentage of the medical profession. It is continuously stressed that the crux of the cancer problem is very early diagnosis and immediate, adequate, and approved treatment; therefore unless the entire medical profession becomes alert to the problem, lay education will be of little benefit and the morbidity and mortality of the disease will continue to mount.

More and more the problem of diagnosis and treatment of malignant disease is developing into a specialty. Diagnostic procedures such as the proper evaluation of the clinical history and physical findings, the biopsy, the correct interpretation of x-ray films, and the precise decision on the form of therapy are problems that can be answered to the best interest of the individual patient only by one well trained in this field. The problem of therapy deserves concrete consideration. Too frequently the surgeon advises operative therapy not because this form of treatment is best suited to the case in question but because it represents his one and formidable type of treatment. On the other hand the radiologist may advise irradiation either by radium or x-ray on the same basis, because he has the facilities for such therapy and it is his means of livelihood and, perhaps, of assisting him in paying for expensive equipment. It is obvious that such trends cannot and do not answer the problem of cancer therapy.

With this perplexing problem acutely in mind it was a great joy to review the recent new work by Pack and Livingston.[2] In this extensive treatise is concentrated the largest amount of concrete and practical information on the subject of cancer therapy that is known to the reviewer. An adequate or just review is impossible in the comparatively limited space allocated for the purpose. This treatise consists of three volumes totaling 2,600 pages prepared by 147 authorities of national and international reputation in their respective fields of cancer diagnosis and therapy under the editorship of two eminently qualified men. The work is illuminated by 1,500 illustrations. Every phase of the problem of cancer therapy is discussed in detail and with such clarity and precision that little if anything is left to the imagination of the reader.

In volume I, one of the outstanding sections is that devoted to general principles of treatment. Here are discussed such subjects as the prevention of cancer, the organization of a tumor clinic, microscopic grading, the surgical, punch and aspiration biopsy, the principles of radiation therapy, and a chapter on radiosensitivity of tumors by Stewart and Farrow that is a classic. This section comprises 274 pages prepared by 18 contributors including such eminent authorities as Ewing, Greenough, Broders, Stewart, Quimby, Pack, Livingston, Chaoul, and others. Volume I also contains descriptions of the treatment of cancer of the mouth and pharynx, larynx, neck, thyroid and parathyroids, breast, and the chest. All of the material is in excellent form and teems with pertinent information; such topics as the treatment of carcinoma of the lip, radiation treatment of cancer of the buccal mucosa, radiation therapy of cancer of the tongue and the treatment of carcinoma of the floor of the mouth are milestones in surgical literature. A most logical and lucid description by Duffy of the indications for neck dissection and for irradiation of cervical nodes in intraoral carcinoma, is ample reason for possessing the treatise. The editors' introduction to the treatment of cancer of the breast is a truly conspicuous contribution.

In volume II are considered the esophagus, stomach, the intestinal tract, colon, rectum, and the female genitals. The thoroughness with which the subjects are presented can be illustrated by a survey of the section on the treatment of cancer of the rectum. Such eminent authorities as Jones, Rankin, Lahey and Cattell, Babcock, Spangler, Burch, and Yeomans are contributors. Binkley and Shedden present the subject of radiation therapy.

Volume III is devoted to the urinary system, male genitals, the skin, eye and orbit, the nervous system, bone and synovial membranes, the lymphoid system, and miscellaneous, the latter including electrosurgical treatment, control of hemorrhage, rectal anesthesia, fever therapy, colloidal lead therapy and

[1] MODERN MEDICAL THERAPY IN GENERAL PRACTICE (in three volumes). Edited by David Preswick Barr, A.B., M.D., LL.D. Baltimore: The Williams & Wilkins Co., 1940.

[2] TREATMENT OF CANCER AND ALLIED DISEASES. By 147 Authorities. Edited by George T. Pack, B.Sc., M.D., F.A.C.S., and Edward M. Livingston, B.Sc., M.D. Vols. 1, 2, 3. New York and London: Paul B. Hoeber, Inc., 1940.

the care of patients in the incurable and terminal stages of cancer. Throughout this volume the same thoroughness and clarity prevail. Subjects so important in correct evaluation as to treatment of cancer of the penis and bladder, kidney tumors, lesions of the skin, and the like, are so clearly classified and the treatment so specifically described that nothing is left for the reader to question.

In résumé it must be conceded that the final word on cancer therapy has not been spoken, however, the advancements made in the past decade are significant, even more, they are auspicious. The recognition of such a fact as cited by Martin relative to cancer of the lip (vol. I, p. 325) that "Early cases with lesions up to 1.5 cm. in diameter should be cured in practically 100 per cent of instances if the patient is regularly observed and examined for recurrences" is, indeed, encouraging and should initiate a profound optimism and stimulate every physician to apply himself to the task of early recognition of the disease and the application of the proper type of therapy for the cancerous lesion.

It is the conviction of the reviewer that this treatise will do more to enlighten the medical profession on the cancer problem, specifically cancer therapy, than any other single effort. No one can read this work without obtaining a boundless enthusiasm intimately interwoven with lucid and concise information. It clearly points the way of cancer therapy into surgical or radiological channels or a manifest combination of the two and establishes the fact that adequate diagnostic and therapeutic procedures for cancer can be conducted only by a harmonious and intelligent co-operation of the pathologist, the radiologist, and the surgeon. This treatise will become the Mecca for the cancer therapist who really desires to give his patients the best type of treatment. JOHN A. WOLFER.

THE two volume text, *Specialties in Medical Practice*,[1] edited by Dr. E. V. Allen of the division of medicine of the Mayo Clinic is published in loose-leaf form by Thomas Nelson and Sons. It is closely patterned after the style of Nelson's *Medicine and Surgery* and is apparently designed to compensate for the special medicine and surgical fields that are not already covered in their regular volumes. Outside of the comparatively new fields of vitamins, endocrinology, and perhaps allergy, these various sections do not present anything particularly new in these specialties but rather offer in a brief form the fundamentals of diagnosis and treatment of the most commonly seen disorders in these special fields. Most of these sections are profusely illustrated with both black and white and colored prints. Some of the sections are summarized in chart form at the end of the discussion.

This text contains sections on ophthalmology, otolaryngology, neurology, psychiatry, vitamins and vitamin deficiency diseases, allergy, orthopedic surgery, endocrinology, urology, proctology, dermatology, and syphilology. The latter, being written by Dr. Svend Lomholt of Copenhagen, Denmark, is still in preparation. These chapters are not arranged in any particular order in these volumes and seem to be rather haphazardly compiled. Chapters are written by men well known in their particular fields of medical and surgical practice. Louis A. Buie, psychology, and John L. Emmett, urology, are the only other contributors to the volume from the Mayo clinic.

Dr. Allen has attempted to present through these men only the most fundamental and practical points regarding their specialties. Rare diseases, etiology, pathology, and descriptions of difficult operative techniques have not been emphasized. Because this book has been designed mainly for the general practitioner so that he can receive practical and useful information that will aid him in his practice, emphasis has been placed on symptoms, diagnosis, and treatment of the most common disorders in these various fields. The style and method of these presentations are, of course, as varied as the number of contributors. Sections on ophthalmology, otolaryngology, endocrinology, vitamin diseases, and urology are particularly well written from the standpoint of simplicity in presentation, usefulness, and therapeutic technique. Other sections, such as those on obstetrics and gynecology, are presented in the style and detail of a regular textbook and seem to be out of place in this type of volume.

The sections on neurology, psychiatry, and orthopedic surgery are too brief and sketchy. It is realized that the scope of these specialties is extremely broad, and one wonders whether or not they lend themselves to contraction. In an attempt to cover the whole field of the specialties in a relatively small section, practical diagnostic points and therapeutic measures that might be used by the general practitioner have not been presented in an adequate manner.

On the whole these volumes are a good supplement to the already established sets of Nelson's *Loose Leaf Medicine and Surgery*. They should be of interest to the busy general practitioner who finds textbooks on these subjects written in a style that is too complicated and in too much detail. It may be of value to the specialist who would like to acquire a speaking acquaintance with the progress that has been made in other fields of medical practice while he has been concentrating in his own special field. It is not a good text for the medical student as it does not give the underlying pathology, basic facts, and reasons for procedure. There is too much superstructure and not enough foundation.

Dr. Allen has done well with the material from many diversified fields in bringing them together in one text, so as to make it possible for the busy general practitioner or specialist to digest these various subjects in small doses at bedtime.

H. A. LINDBERG.

[1] SPECIALTIES IN MEDICAL PRACTICE. Edited by Edgar Van Nuys Allen, M.D. With a Foreword by Donald C. Balfour, M.D. New York and Edinburgh: Thomas Nelson & Sons, 1940.

THE past two or three years have seen the appearance of several books dealing with the subject of craniocerebral injuries—indeed "a major twentieth century problem"—and all those which have been read by this reviewer agree in general in their explanation of the pathology incident to such injury and the proper treatment of the immediate injury. Gross and Ehrlich[1] agree with the apparently prevailing thought that the term "concussion" is a poor one and that the actual changes in the tissues during such a state are unknown, yet they, likewise, fail to supply a term which is satisfactory for this very definite clinical posttraumatic syndrome.

Their diagnostic approach is direct and simple, taking advantage of modern methods but being careful not to overmanipulate the patient. They believe that no rigid plan of treatment can be followed, since the condition of patients with craniocerebral injuries varies widely from individual to individual. They stress the need of attention to simple but important matters such as the care of bowels, bladder, and skin, the adequate nourishment of the patient, and above all sufficient *rest* for the patient. They do not advocate routine lumbar puncture.

A good deal of space is given over to the matter of operative technique, a subject which could have been omitted to allow a broader treatment of posttraumatic convulsive states, psychotic manifestations, and other complications of head injuries, since most surgeons have their own favorite technique of operation in such cases. It is interesting to note that they drain the operative wound after the removal of an extradural clot, but they do not drain the wound after, for example, the repair of a gunshot wound of the scalp, skull, and brain.

The book is short and therefore some of the subjects are necessarily treated in a "telescopic" manner. Chapter XII, on the subject of gunshot wounds, is full of practical information, and it seems particularly timely in this present period of world-wide military strife. There is appended a good bibliography.

JOHN MARTIN.

[1] DIAGNOSIS AND TREATMENT OF HEAD INJURIES. By Sidney W. Gross, M.D., F.A.C.S., and William Ehrlich, M.D. Introduction by Percival Bailey, M.D., Ph.D. New York and London: Paul B. Hoeber, Inc., 1940.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

THE ROCKEFELLER FOUNDATION, ANNUAL REPORT 1939. New York, 1940.

PROBLEMS OF NERVOUS ANATOMY. By J. Boeke, LL.D. (Glasg.). London: Oxford University Press, 1940.

OFFICE CLINICAL CHEMISTRY; A LABORATORY GUIDE FOR THE PRACTITIONER AND HOSPITAL. By Emanuel M. Abrahamson, B.S., Ch.E., M.A., Ph.D., M.D. London, New York, Toronto: Oxford University Press, 1940.

EXPECTANT MOTHERHOOD. By Nicholson J. Eastman, M.D. Boston: Little, Brown & Co., 1940.

HANDBOOK OF SKIN DISEASES. By Leon Hugh Warren, B.A., M.D., M.Sc. (Med.). With a foreword by F. D. Weidman, M.D. New York, London: Paul B. Hoeber, Inc., 1940.

THE ANATOMY OF THE FEMALE PELVIS. By F. A. Maguire, C.M.G., D.S.O., V.D., M.D., Ch.M., F.R.C.S. (Eng.), F.R.A.C.S., F.R.C.O.G. 3d ed. Sydney, London: Angus & Robertson, 1940.

HUGH YOUNG; A SURGEON'S AUTOBIOGRAPHY. New York: Harcourt, Brace & Co., 1940.

CARLOS FINLAY AND YELLOW FEVER. By Carlos F. Finlay, M.D., F.A.C.S., Edited by Morton C. Kahn, M.A., Ph.D., Sc.D. Published under the auspices of The Institute of Tropical Medicine of the University of Havana. New York: Oxford University Press, 1940.

CHAMBERS'S TECHNICAL DICTIONARY. Edited by C. F. Tweney and L. E. C. Hughes, A.C.G.I., D.I.C., B.Sc., Ph.D., A.M.I.E.E., A.M.I.R.E., M.A.S.A. New York: The Macmillan Co., 1940.

A TEXTBOOK OF SURGICAL NURSING. By Henry S. Brookes, Jr., M.D. and Pearl Castile, R.Dn., A.B., M.A. 2d ed. St. Louis: The C. V. Mosby Co., 1940.

MANAGEMENT OF THE CARDIAC PATIENT. By William G. Leaman, Jr., M.D., F.A.C.P. Philadelphia, London, and Montreal: J. B. Lippincott Co., 1940.

NEW AND NONOFFICIAL REMEDIES, 1940. Containing Descriptions of the Articles Which Stand Accepted by the Council on Pharmacy and Chemistry of the American Medical Association on January 1, 1940. Chicago: American Medical Ass., 1940.

ANNUAL REPRINT OF THE REPORTS ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION FOR 1939 WITH THE COMMENTS THAT HAVE APPEARED IN THE JOURNAL. Chicago: American Medical Ass., 1939.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

GEORGE P. MULLER, Philadelphia, *President*
EVARTS A. GRAHAM, St. Louis, *President-Elect*

Committee on Arrangements
JOHN A. WOLFER, *Chairman;* CHARLES B. PUESTOW, *Secretary*

PROGRAM FOR THE CHICAGO CLINICAL CONGRESS

THE 1940 Clinical Congress of the American College of Surgeons will be held in Chicago, October 21–25. This will be the thirtieth assembly of this great organization of surgeons of the United States, Canada, and Latin America, the first of these meetings having been held in Chicago in 1910. The surgeons of this city are privileged to be hosts to this meeting for the eighth time since the Clinical Congress of Surgeons of North America initiated this vast educational program which places the primary emphasis upon well organized clinics conducted in the hospitals of a great medical center. Under the leadership of a strong and representative committee of Chicago surgeons, the clinical program to be presented by the five medical schools and forty approved hospitals will constitute a highly organized postgraduate experience for all those attending the meeting. Representing as it does the interests of all surgical specialists as well as general surgeons, this Congress will provide a complete and varied program during the five days of the meeting.

CLINICAL PROGRAM

The major feature of the Clinical Congress, as arranged by the local committee, will be an extensive schedule of operative and non-operative clinics, demonstrations, symposia, and exhibits conducted by over six hundred Chicago surgeons. These presentations will cover all phases of the clinical activities in the surgical and other departments of the hospitals and medical schools. The latest advances in diagnostic methods, surgical techniques and operative procedures will be demonstrated. Many of the clinics will deal with pertinent subjects related to pre- and postoperative care of the surgical patient. The program of each operative clinic has been planned with the idea of presenting related problems in diagnosis and other special aspects of the management of the surgical condition under consideration. In so far as possible the end results which have been obtained following the specific surgical treatment will be demonstrated.

Non-operative clinics, demonstrations, symposia and exhibits have been planned to cover various special features of the work which is being done in many of the hospitals and medical schools. Again all of the special fields of surgery will be represented in this phase of the clinical program, affording visiting surgeons the opportunity to study at first hand the latest advances in clinical surgery as well as important experimental and research activities of the Chicago institutions.

The program of each institution is being arranged to cover subjects in general surgery, fractures and other traumas, neurosurgery, orthopedic surgery, thoracic surgery, urology, obstetrics and gynecology, ophthalmology, and otorhinolaryngology. The clinical program, organized under this classification, is being so correlated that those attending the Congress will have ample opportunity to devote their time continuously to clinics dealing with subjects related to the specialty in which they are most interested. Clinics and demonstrations will be held on the afternoon of Monday, October 21, and the mornings and afternoons of each of the four succeeding days.

In order to aid the visiting surgeons in selecting the clinics which they desire to attend, the complete details of the clinical program for the following day, arranged according to the foregoing classification of subjects, will be posted each afternoon in the form of bulletins at headquarters in the Stevens Hotel, permitting the advance requisition of clinic tickets. The *Daily Clinical Bulletin* giving this detailed program in printed form will be distributed each following morning during the Congress.

CLINICAL CONGRESS PROGRAM IN BRIEF

(All sessions at the Stevens Hotel except as noted)

Monday, October 21

10:00 Hospital Conference.
1:30 Panel Discussions (5).
2:00 Clinics in Chicago hospitals.
2:00 Hospital Conference.
2:00 Surgical Film Exhibition, General Surgery.
3:00 Assembly of Initiates, followed by reception—Headquarters of College.
3:30 Panel Discussions (5).
8:00 Presidential Meeting and Convocation.

Tuesday, October 22

7:45 Breakfast Conference.
9:00 Clinics in Chicago hospitals.
9:30 Hospital Conference.
9:30 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
10:00 Surgical Film Exhibition, General Surgery.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2).
1:30 Panel Discussions (5).
2:00 Clinics in Chicago hospitals.
2:00 Hospital Conference.
2:00 Symposium on Fractures and Other Traumas.
2:00 Surgical Film Exhibition, General Surgery.
3:30 Panel Discussions (4).
7:00 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
7:30 Hospital Conference.
8:00 Scientific Session, General Surgery.
8:00 Scientific Session, Ophthalmology.
8:00 Scientific Session, Otorhinolaryngology.

Wednesday, October 23

7:45 Breakfast Conference.
9:00 Clinics in Chicago hospitals.
9:30 Hospital Conference.
9:30 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
9:30 State and Provincial Judiciary Committees.
10:00 State and Provincial Executive Committees.
10:30 State and Provincial Credentials Committees and Committees on Applicants.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2).
12:00 Meeting of Board of Governors.
1:30 Panel Discussions (5).
2:00 Clinics in Chicago hospitals.
2:00 Symposium on Cancer.
2:00 Surgical Film Exhibition, General Surgery.
2:30 Hospital Conference.
3:30 Panel Discussions (5).
7:00 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
7:30 Hospital Conference.
8:00 Scientific Session, General Surgery.
8:00 Joint Session, Sections on Ophthalmology and Otolaryngology.

Thursday, October 24

7:45 Breakfast Conference.
9:00 Clinics in Chicago hospitals.
9:30 Hospital Conference.
9:30 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
10:00 Surgical Film Exhibition, General Surgery.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2).
1:30 Annual Meeting, Fellows of the College.
2:00 Clinics in Chicago hospitals.
2:30 Hospital Conference.
3:00 Panel Discussion—Graduate Training in Surgery.
3:30 Panel Discussions (5).
3:30 Surgical Film Exhibition, General Surgery.
3:30 National and Regional Fracture Committees.
7:00 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
8:00 Scientific Session, General Surgery.
8:00 Scientific Session, Ophthalmology.
8:00 Scientific Session, Otorhinolaryngology.

Friday, October 25

9:00 Clinics in Chicago hospitals.
9:30 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
10:00 Surgical Film Exhibition, General Surgery.
2:00 Clinics in Chicago hospitals.
2:00 Group Clinical Conferences (7).
2:00 Surgical Film Exhibition, General Surgery.

EVENING SCIENTIFIC SESSIONS

The opening scientific session of the Clinical Congress will be held on Monday evening in the Ballroom of the Stevens Hotel, when the Presidential Meeting and Convocation will be combined. The new officers of the College will be inaugurated and the 1940 class of initiates received into fellowship. Distinguished surgeons from foreign countries will then be introduced, following which Dr. George P. Muller, of Philadelphia, will deliver the presidential address taking for his subject "The College and American Surgery." "Principles of Colonic Surgery" will be the subject of the sixth annual oration on surgery to be delivered by Dr. Fred W. Rankin, Lexington, Kentucky.

Scientific meetings will be held on Tuesday, Wednesday, and Thursday evenings at the headquarters hotel, when distinguished members of the profession of international prominence will address the assembled guests of the Congress. Complete programs for all sessions will be found in the following pages. The speakers and subjects of these addresses have been carefully selected in order to assure presentations which will be of interest to those who are practicing in all of the special fields of surgery. These programs have been prepared under the direction of the Board of Regents of the College to give consideration to as many of the latest advances in general surgery and the surgical specialties as possible.

PANEL DISCUSSIONS

Panel discussions have met with such decided success at the Clinical Congress and in the sectional meetings of the College that the schedule this year will include an elaboration of this phase of the program as it is arranged for Monday, Tuesday, Wednesday, and Thursday afternoons. Recognized authorities in special fields are cooperating with the College as leaders and collaborators of these panels; and the 34 topics which have been selected cover many pertinent subjects. This type of program permits more informal discussion of the subject than would be possible in the larger meetings. The plan provides that the leader will present his subject within a ten minute period; collaborators will then discuss various phases briefly, after which general discussion from the floor will be encouraged. These sessions have given unusual opportunities to a large group of surgeons to present their viewpoints on various subjects at the Clinical Congress and in the sectional meetings during recent years, and have proven to be exceedingly popular. Therefore it will be necessary to limit the attendance to those who can be comfortably seated in the conference rooms. Tickets will be available each morning at the registration desk for the panels scheduled for that day.

GROUP CLINICAL CONFERENCES

A new feature of considerable interest to all surgeons attending the Congress will be the series of group clinical conferences which is being arranged for Friday afternoon. Seven special fields of surgery have been selected and an important subject of current interest will be briefly presented in each conference. These special fields are: Fractures and other traumas, neurosurgery, obstetrics and gynecology, orthopedic surgery, plastic surgery, thoracic surgery, and urology. The leaders are selecting the subjects which will be presented. They will direct discussions from the floor and visitors attending the meeting will be invited to present any problem with which they may be concerned—one that is related to the special field under discussion. Recognized authorities will endeavor to answer these questions and offer as much helpful advice as possible. Everyone should benefit by participating in consultation conferences of this nature.

SYMPOSIUM ON CANCER

The Cancer Committee of the College has done outstanding work in furthering the development of cancer clinics in hospitals and providing for the registration of cured cases of malignant disease in the cancer archives. The chairman of this committee will give a brief review of these activities of the College in opening the symposium on cancer which will be held Wednesday afternoon. Other subjects of practical interest which will be presented include various problems in the diagnosis and treatment of "Neurogenic Sarcoma" and "Pelvic Cancer." In addition, one speaker will discuss "The Present Status of Carcinogens and Hormones in Cancer Research" and another will lead a discussion on the subject of "Radiation Damage of Tissue and Its Repair." In this symposium the surgeon, the pathologist and the radiologist will discuss important aspects of the cancer problem which are of current interest.

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS

In this symposium, to be held on Tuesday afternoon, the Chairman of the committee will make a brief statement concerning the work which has been done during the past year by the national and regional fracture committees. The scientific papers to be presented at this meeting will emphasize practical subjects dealing with different types of fractures which present special problems in management and treatment. One of the papers will deal particularly with "Undergraduate Education in Fractures and Other Traumas." The program will be of interest to all surgeons who engage in this type of practice and will afford an opportunity for them to learn different viewpoints regarding the handling of many clinical problems.

OPHTHALMOLOGY AND OTOLARYNGOLOGY

A new departure from the proceedings of the Congress in former years is being arranged in ophthalmology and otolaryngology. Separate sessions scheduled for Tuesday and Thursday evenings will include panel discussions and symposia on important subjects in these fields. The leaders and a group of outstanding surgeons will direct the discussions in these meetings so as to cover many phases of a general subject in each of the specialties and a number of different viewpoints will be expressed. On Wednesday evening, there will be a joint session of ophthalmologists and otolaryngologists with a symposium on the important subject "Primary Treatment of Injuries about the Face." An ophthalmologist, an otolaryngologist, a maxillofacial surgeon, and a neurosurgeon will cover all aspects of this subject. This type of program promises to attract wider interest than the usual presentation of formal papers.

Clinics in Chicago hospitals each morning and afternoon for the visiting ophthalmologists and otolaryngologists will demonstrate surgical work

of a wide variety. On Tuesday, Wednesday and Thursday mornings, in addition to the hospital program, there will be clinical conferences for each group of specialists at the headquarters hotel. These have been arranged so that the leader will briefly survey the field for discussion in a ten minute period. The session will then be broken up into small groups limited to twenty each with a separate leader, where there will be an opportunity for everyone to ask questions and participate in the discussions. Each surgeon may select in advance the small section of the meeting which he wishes to attend. Through this plan the general subject will be discussed thoroughly and everyone will profit by the experience and different viewpoints of his colleagues which will be presented. The success of these conferences is assured. The morning and evening meetings will be preceded by the showing of selected motion picture films dealing with ophthalmology and otolaryngology.

ANNUAL MEETING OF THE FELLOWS

The annual meeting of the fellows will be held on Thursday afternoon in the ballroom of the Stevens Hotel beginning at 1:30. The American College of Surgeons has been a potent force which has not only materially raised the professional and ethical standards of surgery, but has also promoted good hospitalization and general improvement in the practice of medicine in the United States and Canada. These activities have received wide recognition by professional groups and the public as well. Each individual fellow of the College has a part in this work and may extend its influence materially in his local community. Hospital standardization alone offers him unlimited opportunity to provide better medical care for his patients in the hospital in which he works through continuous progress in applying the principles of the minimum standard.

The annual meeting of the fellows affords the officers of the College an opportunity to report on the activities of the organization and to receive suggestions from those who have made possible this vast educational program. Reports will be presented of the work which is being done in hospital standardization, graduate training in surgery, cancer clinics, medical service in industry, the Clinical Congress and sectional meetings of the College, also reports of the committees on fractures and other traumas, bone sarcoma, the library and department of literary research, the hall of the art and science of surgery, and medical motion pictures. The finance committee report will also be presented and the work of the State and Provincial, judiciary, credentials, applicants and executive committees will be reviewed. These reports, which are given to the fellows of the American College of Surgeons, will clearly indicate what this great organization is accomplishing. Every fellow attending the Congress will want to be present at this important meeting.

GRADUATE TRAINING IN SURGERY

Following the annual meeting of the fellows on Thursday afternoon, an open forum on graduate training in surgery will be held, with informal discussions from the floor under leadership which should stimulate comments by widely recognized teachers of surgery who will be present. The report of the Committee on Graduate Training in Surgery which is representative of general surgery as well as the surgical specialties will be given by its Chairman. This report summarizes the program which has been recently undertaken by the College and brings up to date statistical information on approved plans of graduate training in surgery in approximately two hundred hospitals of the United States and Canada.

There will be one formal paper at this meeting which presents the role which the hospital without direct medical school connections may play in developing an adequate educational program in surgery. The plan of teaching which can be developed in this type of institution will be discussed in detail. The remainder of the time will be given over to informal discussions from the floor, and the entire program should elicit the interest of all surgeons, hospital executives and educators who are concerned with the future standards of surgery.

ANNUAL MEETINGS OF COMMITTEES

The annual meetings of the State and Provincial Judiciary, Credentials, and Executive Committees will be held on Wednesday morning. These committees have an important function to perform in the activities of the College. The Credentials Committees and the Committees on Applicants provide an organization which constitutes one of the largest and most carefully deliberate accrediting bodies which exist in the medical profession. Through this organization, the standards of fellowship are maintained and each fellow of the College has a definite responsibility in this work. All members of each of these committees are urged to attend these important sessions.

MEETING OF NATIONAL AND REGIONAL FRACTURE COMMITTEES

The meeting of the National and Regional Fracture Committees will be held on Thursday

afternoon, when this large body of surgeons will assemble for a discussion of the activities of the respective groups. Working in co-operation with the American Red Cross, other local organizations and public officials, these committees have exerted great influence in providing improved methods and facilities for the transportation of the injured. There has also been a concerted effort to improve the treatment of fractures in the hospitals of all communities.

ASSEMBLY OF INITIATES

On Monday afternoon in the Auditorium of the American College of Surgeons headquarters at 40 East Erie Street, there will be held the Assembly of Initiates. This meeting will be presided over by Dr. George P. Muller, President of the College, and Dr. Arthur W. Allen, Boston, Vice-Chairman of the Board of Regents. Dr. Bowman C. Crowell and Dr. Malcolm T. MacEachern will discuss "The Program of the American College of Surgeons." Initiates will then recite the Fellowship Pledge and greetings will be extended by Dr. Evarts A. Graham, St. Louis, President-Elect of the College. Closing remarks will be made by Dr. Irvin Abell, Chairman of the Board of Regents. Following the adjournment of this assembly, a reception for the initiates, their wives, friends and fellows will be held by the officers and Regents of the College.

HOSPITAL CONFERENCES

The twenty-third annual hospital standardization conference will open the Clinical Congress with a meeting at the headquarters hotel on Monday morning at 10:00 o'clock. The report of the 1940 hospital standardization survey—official announcement of the list of approved hospitals and hospitals approved for graduate training in surgery—will be made at this session. In addition, there will be a very interesting discussion on "Medical Preparedness in the Case of a War Emergency." The Surgeon General of the United States Navy, Rear Admiral Ross T. McIntire, will give a brief address and E. W. Jones, Superintendent of the Albany Hospital, Albany, New York, will present the plan which has been developed in this hospital for organizing the entire personnel in case of such an emergency.

The afternoon session on Monday will be given over to significant discussions on convalescent care. The subject will be presented from the sociological and economic viewpoints by leading surgeons and others who have had experience with this problem in their respective communities. Fundamental principles in organizing for adequate convalescent care of the surgical patient will be emphasized.

The Tuesday morning session of the hospital conference will include three important panel discussions: "The Control of Surgery," led by Dr. Harold Foss of Danville, Pennsylvania; "The Control of Infections," led by Dr. Frank L. Meleney of New York; and "The Physical and Administrative Hazards of Anesthesia," led by Dr. John S. Lundy of Rochester, Minnesota. These important panels will be of interest to both hospital personnel and surgeons.

The afternoon session will be devoted to a discussion of nursing problems in the hospital. A paper dealing with the "Essentials of Good Nursing Service" will be followed by a panel discussion on "Administration Costs of Nursing in the Hospital." A keenly debated subject, namely "The Accrediting of Schools of Nursing Education," will also be presented. On Tuesday evening a panel round table conference will cover all phases of organization, professional practice and public relations. These sessions will arouse the interest of all hospital visitors, particularly those who are charged with the responsibility of administration in the small hospital.

On Wednesday morning, at a joint session with the American Association of Medical Record Librarians, the following subjects will be discussed: "Adoption of the Standard Nomenclature," "Proposal of a Surgical Nomenclature," "Punch Card System," and "Relationship of the Medical Record Librarian to the Medical Staff." These subjects present pertinent problems in the organization of a medical records department in the hospital. The afternoon session on Wednesday will be devoted to demonstrations and group conferences covering administrative practices and professional procedures held in ten of the leading approved hospitals of the Chicago area. At the evening session the panel discussions will provide a means for presenting specific questions on hospital procedures to a group of leaders in the hospital field.

On Thursday morning the panel discussion will deal principally with the care of tuberculosis patients in general hospitals, the relation of the administration of the hospital to organized medicine, medical staff organization, health service for hospital personnel, and the cost of hospital care. The conference will close on Thursday afternoon with a series of group conferences and demonstrations dealing with administrative and professional practices in leading Chicago institutions.

A new feature has been added to the hospital program this year in the form of breakfast con-

ferences which will be held on Tuesday, Wednesday, and Thursday mornings. These will start promptly at 7:45 and end promptly at 9:15. While breakfast is being served there will be an opportunity for those attending to participate in a free and animated discussion of each subject under consideration. Each morning the speakers will be allowed ten minutes to present their subject, as follows: Tuesday morning, "The Hospital Administrators Program for Self Development"; Wednesday morning, "The Medical Records Librarians Program for Self Development"; Thursday morning, "The Public Relations Program of Your Hospital." Following these brief introductions to the subjects, each visitor attending the conferences will be called upon to give his or her reaction to the matter under discussion. As the attendance at these morning conferences is expected to be very large, arrangements will be made for visitors to register in advance for these sessions.

Perhaps the most important feature of the hospital conference will be the daily consultation service which will be provided. The information desk will be in charge of personnel of the Hospital Standardization Department of the College who will arrange for consultations on hospital problems with the field representatives of the organization and others who will be able to give authoritative information on various subjects. All who attend the hospital conferences are urged to present their particular problem and avail themselves of this service.

PUBLICATION OF PROCEEDINGS

As in former years, the formal papers which are presented at the scientific sessions of the Congress will be published in a special issue of the official journal of the College, SURGERY, GYNECOLOGY, AND OBSTETRICS, in February following the meeting. This is furnished without additional charge to all fellows, junior candidates, and others who register for the Congress as invited guests. The papers which are presented in connection with the Hospital Standardization Conference will be published in subsequent issues of the *Bulletin* of the American College of Surgeons.

AN EXHIBIT BY THE COLLEGE LIBRARY

The Library and Department of Literary Research will maintain a representative at the Clinical Congress for the purpose of outlining to visiting surgeons the services offered by the department and to list any studies which they may desire to undertake. Samples of the reprint collections which are available through the package library service of the College library, bibliographies which have been compiled on specific subjects, and abstracts and translations from various languages will be shown. The exhibit will include charts and displays illustrating the interests of the bibliophile and the ways in which the College library can meet his needs. Visitors are invited to call upon the Librarian to consult with her with respect to their individual needs or their hospital medical library. Visitors are also invited to inspect the library on the upper floor of the John B. Murphy Memorial Building at 40 East Erie St.

ADVANCE REGISTRATION

The hospitals and medical schools of the Chicago area afford accommodations for a large number of visiting surgeons, but to insure against overcrowding, attendance at the Congress will be limited to the number that can be comfortably accommodated at the clinics. The limit of attendance will be based on a survey determining the available facilities in the participating institutions. It is expected, therefore, that surgeons who wish to attend the Congress will register in advance.

A registration fee will be required in order to provide funds with which to meet expenses of the meeting. A formal receipt will be issued to each surgeon registering in advance which will be exchanged for a general admission card upon presentation at headquarters during the Congress. This card, which is not transferable, must accompany all requests for clinic tickets, tickets for the panel discussions and be presented for admission to the scientific sessions.

A resolution adopted by the Board of Regents provides that the registration fee for fellows of the College and endorsed junior candidates shall be $5.00; that no fee for the 1940 Clinical Congress shall be required of initiates (class of 1940); that the fee for surgeons who are not fellows, attending as invited guests of the College, shall be $10.00.

As in previous years, admission to clinics and demonstrations in the hospitals and certain of the scientific meetings at headquarters will be controlled by means of tickets. This plan provides for the distribution of visiting surgeons at the various clinics and other meetings and helps to insure against overcrowding. The number of tickets issued for any clinic or session will be limited to the capacity of the room in which the session is held. Visiting surgeons are urged to cooperate in making the clinic ticket plan a success.

RAILROAD FARES

As no special rates have been authorized by the railroads of the United States or Canada for the 1940 Clinical Congress in Chicago, in accordance with the policy adopted by the railroads, no certificates will be required. However, round trip tickets to Chicago, sold at less than double the regular one-way fare, will be available from points in the United States and Canada. Return limit privileges of such round trip tickets are liberal but are not uniform in all sections of the country. In most instances the return limit is 30 days or more. Surgeons planning to attend the Congress should consult local ticket agents several days in advance of the date of the meeting for complete information as to the fares, routes, stopover privileges, and return limits on round trip tickets.

HEADQUARTERS—TECHNICAL EXHIBITION

Headquarters for the Congress will be established at the Stevens Hotel where there are unusual facilities for accommodating the Congress. All of the public rooms have been reserved for conferences, registration, ticket bureaus, clinic bulletins, executive offices and scientific exhibits. Thus, all activities of the Congress, except the clinical program, will be located under one roof.

The technical exhibition together with the registration desk will be located on the lower floor of the Stevens Hotel in the large exhibition hall. Leading manufacturers of surgical instruments and supplies, sutures, dressings, pharmaceuticals, operating room equipment, x-ray apparatus and hospital equipment of all kinds, as well as publishers of medical books, will be represented in the exhibition. It will provide for the visiting surgeons an opportunity of carefully inspecting the finest modern products of all those industries which are aiding the work of the surgeon and the hospital.

CHICAGO HOTELS AND THEIR RATES

In addition to the headquarters hotel, the Stevens, there are several first-class hotels within short walking distance of headquarters, providing ample hotel facilities at reasonable rates. It is suggested that reservation of hotel accommodations be made at an early date. The following hotels are recommended by the Committee:

	Minimum Rate with Bath	
	Single	Double
Auditorium, 430 S. Michigan Ave	$2.50	$4.00
Bismarck, 171 W. Randolph St	3.50	5.00
Blackstone, Michigan Ave. at 7th St	4.00	6.00
Congress, 500 S. Michigan Ave	3.00	5.00
Drake, Michigan and Lake Shore Drive	4.00	6.00
Harrison, 57 E. Harrison St	2.00	3.00
Lake Shore Drive, 181 Lake Shore Drive	4.00	6.00
LaSalle, 10 N. LaSalle St	2.50	4.00
Morrison, 79 W. Madison St	2.50	4.00
Palmer House, 15 E. Monroe St	3.50	5.00
Sherman, 106 W. Randolph St	2.50	4.00
Stevens, 720 S. Michigan Ave	3.00	4.50

ASSEMBLY OF INITIATES

Monday, 3:00 p.m., Auditorium, American College of Surgeons, 40 East Erie St.

Processional—Initiates, Officers, Regents, and Governors.
Opening Remarks. GEORGE P. MULLER, M.D., Philadelphia, President.
The Program of the American College of Surgeons:
ARTHUR W. ALLEN, M.D., Boston, Vice Chairman, Board of Regents
BOWMAN C. CROWELL, M.D., Chicago, Associate Director.
MALCOLM T. MACEACHERN, M.D., Chicago, Associate Director.
The Fellowship Pledge. Recital by Initiates.
Greetings to the Initiates. EVARTS A. GRAHAM, M.D., St. Louis, President-elect.
Closing Remarks. IRVIN ABELL, M.D., Louisville, Chairman, Board of Regents.
Signing of the Fellowship Roll. The Initiates.
Reception by the officers and regents for the fellows and initiates and members of their families.

PROGRAMS FOR EVENING SESSIONS

Presidential Meeting and Convocation—Monday, 8:00 p.m.—Ballroom, Stevens Hotel

GEORGE P. MULLER, M.D., Philadelphia; President, American College of Surgeons, Presiding.

Processional—Officers, Regents, and Honorary Guests.

Invocation.

Address of Welcome. JOHN A. WOLFER, M.D., Chicago; Chairman, Committee on Arrangements.

Introduction of Foreign Guests. ARTHUR W. ALLEN, M.D., Boston; Vice Chairman, Board of Regents.

Address of Retiring President: The College and American Surgery. GEORGE P. MULLER, M.D., Philadelphia.

Inauguration of Officers:

President: EVARTS A. GRAHAM, M.D., St. Louis.

First Vice President: OLIVER S. WAUGH, M.D., Winnipeg.

Second Vice President: ALBERT O. SINGLETON, M.D., Galveston.

Presentation of Initiates for Fellowship. IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents.

Conferring of Fellowships by the President. EVARTS A. GRAHAM, M.D., St. Louis.

Conferring of Honorary Fellowships. The President.

Medical Records Prize Award.

Annual Oration on Surgery: Principles of Colonic Surgery. FRED W. RANKIN, M.D., Lexington.

Tuesday, 8:00 p.m.—Ballroom, Stevens Hotel

EVARTS A. GRAHAM, M.D., St. Louis; President, American College of Surgeons, Presiding.

Surgical Complications in Pregnancy. JOHN R. FRASER, M.D., Montreal.

Aseptic Resections in the Gastrointestinal Tract, with Special Reference to Resection of the Stomach. OWEN H. WANGENSTEEN, M.D., Minneapolis.

Symposium: The Esophagus.

Observations on the Diagnosis and Treatment of Caustic Burns, Cicatricial Stenosis and Achalasia. GABRIEL TUCKER, M.D., Philadelphia.

Surgical Treatment of Achalasia. ALTON OCHSNER, M.D., and MICHAEL E. DEBAKEY, M.D., New Orleans.

Recent Progress in the Surgical Treatment of Carcinoma of the Esophagus. WILLIAM E. ADAMS, M.D., Chicago.

Wednesday, 8:00 p.m.—Ballroom, Stevens Hotel

OLIVER S. WAUGH, M.D., Winnipeg; Vice President, American College of Surgeons, Presiding.

Sulfonamide Therapy as an Aid to Surgery. JOHN S. LOCKWOOD, M.D., Philadelphia.

Oration on Traumatic Surgery: Treatment of Traumas of Skin and Subcutaneous Tissues. FREDERIC W. BANCROFT, M.D., New York.

The Civilian Surgeon in War. BRIGADIER GENERAL RAYMOND F. METCALFE, M.C., U.S.A., Washington.

A Brief Statement of the Work of the National Research Council Committee on Surgery in Connection with the Program of Military Preparedness. EVARTS A. GRAHAM, M.D., St. Louis.

Thursday, 8:00 p.m.—Ballroom, Stevens Hotel

ALBERT O. SINGLETON, M.D., Galveston; Vice President, American College of Surgeons, Presiding.

Gastrojejunocolic Fistula. DAMON B. PFEIFFER, M.D., Philadelphia.

Some Factors Influencing the Curability of Cancer of the Stomach. THOMAS F. MULLEN, M.D., San Francisco.

The Management of the Neurogenic Bladder in Traumatic Lesions of the Spinal Cord and Cauda Equina. REED M. NESBIT, M.D., Ann Arbor, Mich.

Heparin in Thrombosis and Blood Vessel Surgery. D. W. GORDON MURRAY, M.D., Toronto.

PROGRAMS FOR EVENING SESSIONS

OPHTHALMOLOGY

Tuesday, 8:00 p.m.—Stevens Hotel

Panel Discussion: Surgery of Squint. WILLIAM THORNWALL DAVIS, M.D., Washington, Presiding.
Collaborators: F. BRUCE FRALICK, M.D., Ann Arbor, Mich.; SANFORD R. GIFFORD, M.D., Chicago.

Thursday, 8:00 p.m.—Stevens Hotel

SANFORD R. GIFFORD, M.D., Chicago; Chairman, Advisory Council for Ophthalmology, Presiding.

Symposium: Advances of the Past Ten Years in Ophthalmic Surgery.
Principles of Cataract Surgery. JOHN H. DUNNINGTON, M.D., New York.
Glaucoma Surgery. ALGERNON B. REESE, M.D., New York.
Retinal Detachment Surgery. DERRICK VAIL, M.D., Cincinnati.

OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

Wednesday, 8:00 p.m.—Stevens Hotel

HARRY S. GRADLE, M.D., Chicago; Regent, American College of Surgeons, Presiding.

Symposium: Treatment of Injuries about the Face.
The Treatment of Injuries of the Globe and the Lids, and the Orbit. EDMUND B. SPAETH, M.D., Philadelphia.
Primary Care of Injuries of the Face and Jaws. V. H. KAZANJIAN, M.D., Boston.
The Late Care of Severe Injuries of the Face and Jaws. EARL C. PADGETT, M.D., Kansas City, Mo.
Neurosurgical Aspects of Facial Injury. HARRY H. KERR, M.D., Washington.

OTORHINOLARYNGOLOGY

Tuesday, 8:00 p.m.—Stevens Hotel

SAMUEL SALINGER, M.D., Chicago; Member, Executive Committee on Arrangements, Presiding.

Symposium: Ménière's Disease.
Medical Treatment of Ménière's Disease. HAROLD G. TOBEY, M.D., Boston.
The Use of Histamine in Ménière's Disease. BAYARD T. HORTON, M.D., Rochester, Minn.
The Surgical Treatment of Ménière's Disease. WALTER E. DANDY, M.D., Baltimore.
General Discussion.

Thursday, 8:00 p.m.—Stevens Hotel

GORDON B. NEW, M.D., Rochester, Minn.; Member, Advisory Council for Otorhinolaryngology, Presiding.

Body Section Roentgenography in Relation to Diagnosis in Diseases of the Nasal Accessory Sinuses. SHERWOOD MOORE, M.D., and ALFRED J. CONE, M.D., St. Louis.

Tumors of the Upper Jaw, Particularly in Relation to the Sinuses. CARL W. WALDRON, M.D., Minneapolis.

History and Present Status of Operations on the Labyrinthine Capsule for Otosclerosis. SAMUEL J. KOPETZKY, M.D., New York.

GROUP CLINICAL CONFERENCES

GENERAL SURGERY AND SURGICAL SPECIALTIES

Friday, 2:00 p.m.—Stevens Hotel

Treatment of Compound Fractures. GUY A. CALDWELL, M.D., New Orleans, Presiding.
General Review. GUY A. CALDWELL, M.D., New Orleans.
First Aid and Transportation Splinting. J. E. M. THOMSON, M.D., Lincoln, Neb.
Debridement and Operative Treatment. J. ALBERT KEY, M.D., St. Louis.
Methods of Splinting and Postoperative Treatment. HAROLD R. CONN, M.D., Akron, Ohio.

Orthopedic Surgery. ARTHUR STEINDLER, M.D., Iowa City, Presiding.
Open forum.

Diagnosis and Treatment of Intrathoracic Tumors. FRANK S. DOLLEY, M.D., Los Angeles, Presiding.
Mediastinal Tumors Amenable to Surgical Removal. FRANK S. DOLLEY, M.D., Los Angeles.
Bronchogenic Tumors. ALTON OCHSNER, M.D., New Orleans.
Bronchoscopic Consideration. CHEVALIER L. JACKSON, M.D., Philadelphia.
Operative Technique. EVARTS A. GRAHAM, M.D., St. Louis.
Open forum. JOHN ALEXANDER, M.D., Ann Arbor, Mich., Leader.

Major Trigeminal Neuralgia. FRANCIS C. GRANT, M.D., Philadelphia, Presiding.
General Discussion. FRANCIS C. GRANT, M.D., Philadelphia.
Importance of Alcohol Injection in the Treatment of Major Trigeminal Neuralgia. GILBERT HORRAX, M.D., Boston.
Total versus Subtotal Section of the Sensory Root. WINCHELL MCK. CRAIG, M.D., Rochester, Minn.
Operative Methods in Major Trigeminal Neuralgia. W. JAMES GARDNER, M.D., Cleveland.
Sensory Changes and the Prevention of Ocular and Other Complications Following Operation on the Sensory Root. LOYAL DAVIS, M.D., Chicago.

Malignancies of the Kidney and Ureter. T. LEON HOWARD, M.D., Denver, Presiding.
Wilms' Tumors in Children. MEREDITH F. CAMPBELL, M.D., New York.
Hypernephroma. REED M. NESBIT, M.D., Ann Arbor, Mich.
Malignancies of the Ureter. VINCENT J. O'CONOR, M.D., Chicago.
Papillary Carcinomas of the Kidney. W. CALHOUN STIRLING, M.D., Washington.

Deformities of the Ear, Congenital and Acquired. H. L. D. KIRKHAM, M.D., Houston, Texas, Presiding.
Development of the Ear and Some Remarks on Lop Ears. DONALD W. MACCOLLUM, M.D., Boston.
The Physic Phases of Ear Deformities. VILRAY P. BLAIR, M.D., St. Louis.
Lop Ears and Conditions Associated with Congenital Deformities. GORDON B. NEW, M.D., Rochester.
Reconstruction of the Ear. GEORGE WARREN PIERCE, M.D., San Francisco; FERRIS SMITH, M.D., Grand Rapids, Mich.
Use of Preserved Ear Cartilage in Ear Reconstruction. H. L. D. KIRKHAM, M.D., Houston, Texas.

The Management of Uterine Prolapse. LOUIS E. PHANEUF, M.D., Boston, Presiding.
The Anatomical Supports of the Uterus and Pelvic Viscera. FREDERICK H. FALLS, M.D., Chicago.
The Interposition Operation (Watkins) in the Management of Uterine Prolapse. HILLIARD E. MILLER, M.D., New Orleans.
Vaginal Hysterectomy in the Management of Uterine Prolapse. ROBERT A. ROSS, M.D., Durham, N.C.
The Manchester Operation (Donald-Fothergill) in the Management of Uterine Prolapse. JOHN R. FRASER, M.D., Montreal.
Hernia of Douglas' Culdesac Complicating Uterine Prolapse. GEORGE GRAY WARD, M.D., New York.

OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

Tuesday, 11:00 a.m.—Stevens Hotel

Surgical Indications in Glaucoma. HARRY S. GRADLE, M.D., Chicago, Presiding.

Collaborators: WALTER S. ATKINSON, Watertown, N.Y.; EUGENE M. BLAKE, M.D., New Haven; VIRGIL G. CASTEN, M.D., Boston; CARL FISHER, M.D., Los Angeles; JONAS S. FRIEDENWALD, M.D., Baltimore; EVERETT L. GOAR, M.D., Houston, Texas; LOUIS S. GREENE, M.D., Washington; HENRY C. HADEN, M.D., Houston, Texas; SAMUEL J. MEYER, M.D., Chicago; CLARENCE A. VEASEY, M.D., Spokane, Wash.

Rôle of Chemotherapy in Suppurative Diseases of the Middle Ear. JOHN J. SHEA, M.D., Memphis, Tenn., Presiding.

Collaborators: ALBERT L. BASS, M.D., Louisville; JOSEPH C. BECK, M.D., Chicago; JAMES A. FLYNN, M.D., Washington; WILLIAM D. GILL, M.D., San Antonio; GORDON F. HARKNESS, M.D., Davenport; FREDERICK T. HILL, M.D., Waterville, Me.; ROY H. PARKINSON, M.D., San Francisco; COLVIN C. PERDUE, M.D., Mobile; CLARENCE H. SMITH, M.D., New York.

Wednesday, 11:00 a.m.—Stevens Hotel

Surgical Management of Injuries to the Eye. ARTHUR M. CULLER, M.D., Dayton, Ohio, Presiding.

Collaborators: LAWRENCE G. DUNLAP, M.D., Anaconda, Mont.; ERLING W. HANSEN, M.D., Minneapolis; FREDERICK A. KIEHLE, M.D., Portland, Ore.; DANIEL B. KIRBY, M.D., New York; FRANK N. KNAPP, M.D., Duluth; M. PAUL MOTTO, M.D., Cleveland; BRITTAIN FORD PAYNE, M.D., New York; LYLE S. POWELL, M.D., Lawrence, Kans.; HARVEY E. THORPE, M.D., Pittsburgh; JOHN O. WETZEL, M.D., Lansing, Mich.

Indications for Surgery in Sinus Disease. SAMUEL IGLAUER, M.D., Cincinnati, Presiding.

Collaborators: HOWARD C. BALLENGER, M.D., Winnetka, Ill.; MATTHEW S. ERSNER, M.D., Philadelphia; ROBERT H. FRASER, M.D., Battle Creek, Mich.; C. H. MCCASKEY, M.D., Indianapolis; SAMUEL SALINGER, M.D., Chicago; W. LIKELY SIMPSON, M.D., Memphis, Tenn.

Thursday, 11:00 a.m.—Stevens Hotel

Minor Surgery in Ophthalmology. MEYER WIENER, M.D., St. Louis, Presiding.

Collaborators: CLYDE A. CLAPP, M.D., Baltimore; RAY K. DAILY, M.D., Houston, Texas; THEODORE J. DIMITY, M.D., New Orleans; JAMES N. GREEAR, JR., M.D., Washington; WENDELL L. HUGHES, M.D., Hempstead, N.Y.; VINCENT L. JONES, M.D., St. Louis; CLYDE E. MCDANNALD, M.D., New York; LYLE S. POWELL, M.D., Lawrence, Kans.; RALPH O. RYCHENER, M.D., Memphis, Tenn.; WALTER STEVENSON, M.D., Quincy, Ill.

Osteomyelitis of the Skull Bones (Sinus and Mastoid Complications). THOMAS E. CARMODY, M.D., Denver, Presiding.

Collaborators: WILLIAM F. CLEVENGER, M.D., Indianapolis; DANIEL S. CUNNING, M.D., New York; JOSEPH E. J. KING, M.D., New York; FRANCIS L. LEDERER, M.D., Chicago; EDWARD A. LOOPER, M.D., Baltimore; WILLIAM J. MELLINGER, M.D., Santa Barbara, Calif.; ABRAHAM O. WILENSKY, M.D., New York.

GRADUATE TRAINING IN SURGERY

Thursday, 3:00 p.m.—Ballroom, Stevens Hotel

DALLAS B. PHEMISTER, M.D., Chicago; Chairman, Committee on Graduate Training for Surgery, Presiding.

The Rôle in a Teaching Program for Graduate Training in Surgery of a Hospital without University Connections. CHARLES A. BOWERS, M.D., Cleveland.

Open forum: Graduate Training in General Surgery and the Surgical Specialties. ROBIN C. BUERKI, M.D., Madison, Wis., Leader.

Duration of training and objective. Organization and supervision. Basic medical sciences. Clinical material. Organized study.

PANEL DISCUSSIONS

Monday, 1:30 to 3:00 p.m.—Stevens Hotel

Treatment of Acute Cholecystitis. JOHN L. ATLEE, JR., M.D., Lancaster, Pa., Presiding.
Collaborators: FREDERICK E. KREDEL, M.D., Charleston, S. C.; KENT W. BARBER, M.D., Quincy, Ill.; J. LOUIS RANSOHOFF, M.D., Cincinnati.

Dislocation of the Intervertebral Disc. FRANK D. DICKSON, M.D., Kansas City, Mo., Presiding.
Collaborators: W. JASON MIXTER, M.D., Boston; LEWIS J. POLLOCK, M.D., Chicago; EDGAR F. FINCHER, M.D., Atlanta, Ga.

The Treatment of Endocrine Sterility in Woman. EDWIN C. HAMBLEN, M.D., Durham, N. C., Presiding.
Collaborators: JOHN C. BURCH, M.D., Nashville; CONRAD G. COLLINS, M.D., New Orleans.

Complications of Thyroid Surgery. FRANK H. LAHEY, M.D., Boston, Presiding.
Collaborators: HAROLD L. FOSS, M.D., Danville, Pa.; S. L. LEDBETTER, JR., M.D., Birmingham; H. H. SEARLS M.D., San Francisco.

Place of Irradiation Treatment in Cancer of the Breast. E. P. PENDERGRASS, M.D., Philadelphia, Presiding.
Collaborators: FRANK E. ADAIR, M.D., New York; ALSON R. KILGORE, M.D., San Francisco; JAMES F. KELLY, M.D., Omaha; STUART W. HARRINGTON, M.D., Rochester, Minn.

Monday, 3:30 to 5:00 p.m.—Stevens Hotel

Abdominal Traumas. FREDERICK A. BESLEY, M.D., Waukegan, Presiding.
Collaborators: JOHN H. MULHOLLAND, M.D., New York; FRED W. BAILEY, M.D., St. Louis; AMBROSE H. STORCK, M.D., New Orleans.

Carcinoma of the Colon. FREDERICK A. COLLER, M.D., Ann Arbor, Mich., Presiding.
Collaborators: VERNON C. DAVID, M.D., Chicago; CLAUDE F. DIXON, M.D., Rochester, Minn.; THOMAS E. JONES, M.D., Cleveland.

Indications for Cesarean Section. S. A. COSGROVE, M.D., Jersey City, N. J., Presiding.
Collaborators: JAMES RAGLAN MILLER, M.D., Hartford; H. HUDNALL WARE, JR., M.D., Richmond.

Management of Acute Perforated Appendicitis. VERNE C. HUNT, M.D., Los Angeles, Presiding.
Collaborators: LAWRENCE S. FALLIS, M.D., Detroit; WILLIS D. GATCH, M.D., Indianapolis; HENRY K. RANSOM, M.D., Ann Arbor, Mich.

Subacromial Bursitis. HENRY W. MEYERDING, M.D., Rochester, Minn., Presiding.
Collaborators: EARL D. MCBRIDE, M.D., Oklahoma City; CLAY RAY MURRAY, M.D., New York; R. H. FREYBERG, M.D., Ann Arbor, Mich.

Tuesday, 1:30 to 3:00 p.m.—Stevens Hotel

Atypical Giant Cell Tumors of Bone. BRADLEY L. COLEY, M.D., New York, Presiding.
Collaborator: DALLAS B. PHEMISTER, M.D., Chicago.

Nutritional State of the Patient (Vitamins, Proteins). L. KRAEER FERGUSON, M.D., Philadelphia, Presiding.
Collaborators: JOHN B. HARTZELL, M.D., Detroit; A. C. IVY, M.D., Chicago; CHARLES C. LUND, M.D., Boston.

Management of Infections of the Urinary Tract. CHARLES C. HIGGINS, M.D., Cleveland, Presiding.
Collaborators: HENRY O. MERTZ, M.D., Indianapolis; JOHN K. ORMOND, M.D., Detroit; IRA R. SISK, M.D., Madison, Wis.

Acute Abdominal Surgery in Children. THOMAS H. LANMAN, M.D., Boston, Presiding.
Collaborators: EDWARD J. DONOVAN, M.D., New York; WALTER ESTELL LEE, M.D., Philadelphia; W. J. POTTS, M.D., Oak Park, Ill.

Tumors of the Salivary Glands. ALBERT O. SINGLETON, M.D., Galveston, Presiding.
Collaborators: EDWIN I. BARTLETT, M.D., San Francisco; FREDERICK A. FIGI, M.D., Rochester, Minn.; R. B. MALCOLM, M.D., Chicago.

Tuesday, 3:30 to 5:00 p.m.—Stevens Hotel

Surgery in Pulmonary Tuberculosis. LEO ELOESSER, M.D., San Francisco, Presiding.
Collaborators: CASPER F. HEGNER, M.D., Denver; RICHARD H. MEADE, JR., M.D., Philadelphia; VICTOR STRONG RANDOLPH, M.D., Phoenix, Ariz.

Early Recognition of Cancer of the Uterine Cervix and of the Corpus. WILLIAM P. HEALY, M.D., New York, Presiding.
Collaborators: NORMAN F. MILLER, M.D., Ann Arbor, Mich.; FRED J. TAUSSIG, M.D., St. Louis; RAYMOND E. WATKINS, M.D., Portland, Ore.

Intestinal Obstruction. THOMAS G. ORR, M.D., Kansas City, Mo., Presiding.
Collaborators: M. J. HENRY, M.D., Louisville; CHARLES G. JOHNSTON, M.D., Detroit; MIMS GAGE, M.D., New Orleans.

Surgical Diseases of the Pancreas. ALLEN O. WHIPPLE, M.D., New York, Presiding.
Collaborators: ALEXANDER BRUNSCHWIG, M.D., Chicago; JOHN MILTON MCCAUGHAN, M.D., St. Louis.

Wednesday, 1:30 to 3:00 p.m.—Stevens Hotel

Tumors of the Urinary Bladder. HENRY G. BUGBEE, M.D., New York, Presiding.
Collaborators: LOUIS M. ORR, M.D., Orlando, Fla.; CARL RUSCHE, M.D., Los Angeles.

Etiology and Treatment of Gastric Hemorrhage. ROSCOE R. GRAHAM, M.D., Toronto, Presiding.
Collaborators: HOWARD M. CLUTE, M.D., Boston; J. WILLIAM HINTON, M.D., New York; J. SHELTON HORSLEY, M.D., Richmond.

Anesthesia in Cranial Surgery. GILBERT HORRAX, M.D., Boston, Presiding.
Collaborators: MAX MINOR PEET, M.D., Ann Arbor, Mich.; JOHN S. LUNDY, M.D., Rochester, Minn.

Healing of Wounds. SUMNER L. KOCH, M.D., Chicago, Presiding.
Collaborators: LAWRENCE CHAFFIN, M.D., Los Angeles; R. ARNOLD GRISWOLD, M.D., Louisville; EDWARD L. HOWES, M.D., Washington.

Liver Tests and Liver Function in Relation to Biliary Surgery. I. S. RAVDIN, M.D., Philadelphia, Presiding.
Collaborators: CHARLES B. PUESTOW, M.D., Chicago; HOWARD K. GRAY, M.D., Rochester, Minn.; HENRY W. CAVE, M.D., New York.

Wednesday, 3:30 to 5:00 p.m.—Stevens Hotel

Penetrating Wounds of the Brain. C. C. COLEMAN, M.D., Richmond, Presiding.
Collaborators: WALLACE B. HAMBY, M.D., Buffalo; HOWARD C. NAFFZIGER, M.D., San Francisco; BYRON STOOKEY, M.D., New York.

Colles' Fracture. PAUL B. MAGNUSON, M.D., Chicago, Presiding.
Collaborators: ROBERT H. KENNEDY, M.D., New York; ROBERT D. SCHROCK, M.D., Omaha; GEORGE E. WILSON, M.D., Toronto.

Infections of the Hand. MICHAEL L. MASON, M.D., Chicago, Presiding.
Collaborators: HOMER D. DUDLEY, M.D., Seattle; HENRY C. MARBLE, M.D., Boston; JAMES M. WINFIELD, M.D., Detroit.

Surgical Bacteriology. FRANK L. MELENEY, M.D., New York, Presiding.
Collaborators: J. DERYL HART, M.D., Durham, N. C.; EDMUND ANDREWS, M.D., Bloomington, Ill.; WILLIAM A. ALTEMEIER, M.D., Cincinnati.

Immediate Treatment of Burns. GROVER C. PENBERTHY, M.D., Detroit, Presiding.
Collaborators: HARRY C. HULL, M.D., Baltimore; EDMUND BUTLER, M.D., San Francisco; DONALD B. WELLS, M.D., Hartford.
Motion Picture: Final Results Obtained in the Free Skin Grafting of Burns. JAMES BARRETT BROWN, M.D., and FRANK MCDOWELL, M.D., St. Louis.

Thursday, 3:30 to 5:00 p.m.—Stevens Hotel

Anesthesia, Analgesia, and Amnesia in Obstetrics. LOUIS H. DOUGLASS, M.D., Baltimore, Presiding.
Collaborators: FREDERIC SCHREIBER, M.D., Detroit; PAUL TITUS, M.D., Pittsburgh; RALPH M. TOVELL, M.D., Hartford.

Non-malignant Diseases of the Intestines. THOMAS E. JONES, M.D., Cleveland, Presiding.
Collaborators: IRVIN ABELL, M.D., Louisville; J. L. MCGEHEE, M.D., Memphis; CHARLES W. MAYO, M.D., Rochester, Minn.

Anesthesia in Abdominal Surgery. ERWIN R. SCHMIDT, M.D., Madison, Wis., Presiding.
Collaborators: VIRGINIA APGAR, M.D., New York; CHARLES J. BETLACH, M.D., Chicago; F. W. HARTMAN, M.D., Detroit.

Suppurative Conditions of the Lung. NORMAN S. SHENSTONE, M.D., Toronto, Presiding.
Collaborators: WILLIAM F. RIENHOFF, JR., M.D., Baltimore; WILLIAM A. HUDSON, M.D., Detroit; PAUL C. SAMSON, M.D., Oakland, Calif.

Management of Gangrene in Diabetics. ARTHUR A. ZIEROLD, M.D., Minneapolis, Presiding.
Collaborators: WALTER H. NADLER, M.D., Chicago; JOSEPH B. PRIESTLEY, M.D., Des Moines; BEVERLY CHEW SMITH, M.D., New York.

PROGRAMS FOR AFTERNOON SESSIONS

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS

Tuesday, 2:00 p.m.—Stevens Hotel

ROBERT H. KENNEDY, M.D., New York; Chairman, Committee on Fractures and Other Traumas, Presiding.

Results of Fractures of the Tibia and Fibula Treated by Skeletal Traction and Braun-Böhler Splint. HUBLEY R. OWEN, M.D., Philadelphia.

The Recognition and Early Treatment of Injuries of the Genito-Urinary Tract. ALEXANDER HAMILTON PEACOCK, M.D., Seattle, Wash.

Fractures Due to Muscular Violence. FRANK P. STRICKLER, M.D., Louisville.

Undergraduate Education in Fractures and Other Traumas. CLAY RAY MURRAY, M.D, New York.

Extradural Hemorrhage Following Trauma. J. DERYL HART, M.D., and BARNES WOODHALL, M.D., Durham, N. C.

Fractures Involving the Ankle Joint. RALPH G. CAROTHERS, M.D., Cincinnati.

SYMPOSIUM ON CANCER

Wednesday, 2:00 p.m.—Stevens Hotel

FRANK E. ADAIR, M.D., New York; Chairman, Cancer Committee, Presiding.

Neurogenic Sarcoma. DONALD V. TRUEBLOOD, M.D., Seattle.

Present Status of Carcinogens and Hormones in Cancer Research. JOHN J. MORTON, M.D., Rochester, N.Y.

Radiation Damage to Tissue and Its Repair. ERNEST M. DALAND, M.D., Boston.

The Present Status of the Treatment of Gynecologic Cancer with Especial Reference to Results Obtained by Roentgenotherapy. GEORGE KAMPERMAN, M.D., Detroit.

COMMITTEE ON ARRANGEMENTS

EXECUTIVE COMMITTEE

John A. Wolfer, Chairman
Charles B. Puestow, Secretary
Fred L. Adair
William E. Adams
William J. Baker
Frederic A. Besley
Alexander Brunschwig
Fremont A. Chandler
Warren H. Cole
Vernon C. David
Marshall Davison
Harry S. Gradle
Michael L. Mason
Oscar E. Nadeau
Eric Oldberg
Samuel Salinger
Charles F. Sawyer
Kellogg Speed

HOSPITALS AND REPRESENTATIVES

Alexian Brothers—George L. Apfelbach.
Augustana—Oscar E. Nadeau.
Albert Merritt Billings and Bobs Roberts Memorial—Dallas B. Phemister.
Chicago Lying-in—Fred L. Adair.
Chicago Memorial—Peter S. Clark.
Children's Memorial—Albert H. Montgomery.
Columbus—Daniel A. Orth.
Cook County—Marshall Davison.
Evangelical—G. Henry Mundt.
Evanston—Frederick Christopher.
Grant—A. G. Zimmerman.
Henrotin—John A. Graham.
Holy Cross—John F. Ruzic.
Illinois Eye and Ear Infirmary—Thomas D. Allen.
Illinois Masonic—Walter C. Bornemeier.
Jackson Park—Arrie Bamberger.
Loretto—James A. Valentine.
Mercy—Charles F. Sawyer.
Michael Reese—Ralph B. Bettman.
Mother Cabrini—Eugene J. Chesrow.
Mount Sinai—Jacob M. Mora.
Municipal Tuberculosis Sanitarium—Richard Davison.
Norwegian-American—Harold A. Sofield.
Passavant Memorial—Loyal Davis.
Presbyterian—Vernon C. David.
Ravenswood—Darwin B. Pond.
Research and Educational—Warren H. Cole.
St. Anne's—George F. Thompson.
St. Anthony de Padua—Frederick W. Slobe.
St. Bernard's—John G. Frost.
St. Elizabeth's—Martin G. Luken.
St. Joseph—Hugh McKenna.
St. Luke's—Selim W. McArthur.
St. Mary of Nazareth—George M. Mueller.
Shriners' for Crippled Children—Beveridge H. Moore.
Swedish Covenant—Karl L. Vehe.
Veterans Administration—Paul F. Brown.
Washington Boulevard—Arthur R. Metz.
Wesley Memorial—Raymond W. McNealy.
Women and Children's—Maude H. Winnett.

ANNUAL HOSPITAL STANDARDIZATION CONFERENCE

Monday, 10:00 a.m.—Ballroom, Stevens Hotel

GEORGE P. MULLER, M.D., Philadelphia; President, American College of Surgeons, presiding.

Address of the President—The Hospital Program of the American College of Surgeons.

Report of the 1940 Hospital Standardization Survey—Official Announcement of the List of Approved Hospitals. IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents, American College of Surgeons.

Report on Graduate Training in Surgery. DALLAS B. PHEMISTER, M.D., Chicago.

The Hospital of Tomorrow. REV. A. M. SCHWITALLA, S.J., St. Louis; President, Catholic Hospital Association.

Medical Preparedness for National Emergency. ROSS T. MCINTIRE, M.D., Washington; Surgeon-General, United States Navy.

Preparedness for National Emergency from the Standpoint of the Hospital. E. W. JONES, Albany, N. Y.

The Effect of the Present Trend in Specialization in Medicine on Hospital Administration and Service. BENJAMIN W. BLACK, M.D., Oakland, Calif.; President, American Hospital Association.

Discussion. Led by EVARTS A. GRAHAM, M.D., St. Louis; President-Elect, American College of Surgeons.

Monday, 2:00 p.m.—Ballroom, Stevens Hotel

HARRY E. MOCK, M.D., Chicago, presiding.

Convalescent Care—The Missing Link in the Cure of Many Patients. HARRY E. MOCK, M.D., Chicago.

Socio-Economic Aspects of Convalescent Care. ELIZABETH G. GARDINER, New York.

Minimum Standards for Convalescent Hospitals. E. H. L. CORWIN, Ph.D., New York.

Essentials in Planning of Institutions for Convalescent Care. WILLIAM H. WALSH, M.D., Chicago.

Institutional Convalescent Care for Various Types of Patients:

- The Surgical Patient. DONALD B. WELLS, M.D., Hartford, Conn.
- The Medical Patient. NEWELL C. GILBERT, M.D., Chicago.
- The Cancer Patient. FRANK E. ADAIR, M.D., New York.
- The Fracture Patient. ROBERT H. KENNEDY, M.D., New York.
- The Orthopedic Patient. FREDERICK C. KIDNER, M.D., Detroit.
- The Pediatric Patient. LOUIS A. SCHWARTZ, M.D., Detroit.

General discussion. Opened by KARL A. MEYER, M.D., Chicago; JOHN S. COULTER, M.D., Chicago; JOSEPH KING, Chicago.

Monday, 4:30 p.m.—Lounge, 2nd Floor, Stevens Hotel

Consultation Service: Administrative practices and policies; medical staff problems and medical records; pathological service; business management; patients' library service; hospital standardization problems.

Tuesday, 7:45 a.m.—Private Dining Room, Stevens Hotel

Breakfast Conference, Sponsored by the American College of Hospital Administrators in co-operation with the American College of Surgeons: The Hospital Administrators' Program for Self Development. ARTHUR C. BACHMEYER, M.D., Chicago, presiding.

Discussion from the following aspects:

- Institutes for Hospital Administrators. BENJAMIN W. BLACK, M.D., Oakland, Calif.
- Apprenticeship Training. CLAUDE W. MUNGER, M.D., New York.
- Self Study and Personal Development. JOSEPH G. NORBY, Milwaukee.

General discussion. Opened by FLORENCE KING, St. Louis.

Tuesday, 9:30 a.m.—Upper Tower Ballroom, Stevens Hotel

GUY M. HANNER, Colorado Springs, Colo., presiding.

Greetings from the American Protestant Hospital Association. GUY M. HANNER, Colorado Springs, Colo.

Panel Discussions:

- Qualifications for Performing Major Surgery. Conducted by HAROLD L. FOSS, M.D., Danville, Pa.
- Discussion from the standpoints of:
  - The Practical Application of a Plan for the Control of Major Surgery in an Open Hospital. E. MACDONALD STANTON, M.D., Schenectady, N. Y.
  - Organization of the Medical Staff in Relation to the Control of Major Surgery. CLARENCE E. REES, M.D., San Diego, Calif.
- The Respective Rôles of the Professional and Administrative Staffs in the Control of Postoperative Infections. Conducted by FRANK L. MELENEY, M.D., New York.
- Discussion from the standpoints of:
  - Bacteriologist. WILLIAM F. PETERSEN, M.D., Chicago.
  - Operating Room Supervisor. MARGARET K. SCHAFER, Ann Arbor, Mich.
- Anesthesia Hazards. Conducted by JOHN S. LUNDY, M.D., Rochester, Minn.
- Discussion from the standpoints of:
  - Physical Hazards—Explosibility and Inflammability. RALPH M. TOVELL, M.D., Hartford, Conn.
  - Administrative Hazards—The Administration of Anesthesia by Inexperienced Persons. HENRY S. RUTH, M.D., Merion, Pa.

Tuesday, 2:00 p.m.—Upper Tower Ballroom, Stevens Hotel

Conference on Nursing Service and Nursing Education. NELLIE X. HAWKINSON, R.N., Chicago, presiding.

Essentials of Good Nursing Care of the Patient. SISTER M. BERENICE, R.N., Milwaukee.

Panel Discussions:

- Administrative Costs of Nursing Based on Recent Study and Published Report. CLAUDE W. MUNGER, M.D., New York.
- Discussion from the viewpoints of:
  - Special Problems in the Separation of Nursing Education and Nursing Costs. CHARLES A. ROVETTA, Chicago.
  - The Responsibility and Contribution of the Nursing Administrator in a Nursing Cost Study. BLANCHE PFEFFERKORN, R.N., New York.
  - Accounting in Nursing Costs. C. RUFUS ROREM, Ph.D., Chicago.
- The General Plan for Accrediting Schools of Nursing. ELIZABETH C. BURGESS, R.N., New York.
- Discussion from the viewpoints of:
  - Nursing Education. GEORGE A. WORKS, Chicago.
  - Nursing Administration. MARGARET CARRINGTON, R.N., Chicago.
  - Hospital Administration. ROBIN C. BUERKI, M.D., Madison, Wis.

Tuesday, 4:30 p.m.—Lounge, 2nd Floor, Stevens Hotel

Consultation Service: Administrative practices and policies; operating room management; small hospital problems; public relations; outpatient department management; hospital standardization problems.

Tuesday, 7:00 p.m.—Upper Tower Ballroom, Stevens Hotel

Motion pictures.

Panel Round Table Conference: Organization, Management, and Special Problems of the Small Hospital. A. F. BRANTON, M.D., Willmar, Minn., presiding.

Personnel—Securing and Maintaining Competent Administrative and Other Personnel. MARJORIE M. IBSEN, R.N., Highland Park, Ill.

Medical Staff—Organizing and Functioning of the Medical Staff. F. JANE GRAVES, R.N., Alton, Ill.

Adjunct Services—Providing Adequate Laboratory, X-ray, and Other Essential Services for the Treatment of the Patient. F. P. MCNAMARA, M.D., Dubuque, Iowa.

Medical Records—Securing Good Medical Records. ALPHONSUS M. MCCARTHY, M.D., Electric Mills, Miss.

Nursing—Providing Efficient Nursing Service. MABEL L. KUEBLER, R.N., Louisville.

Financing—Meeting the Problem of Finance. VICTOR S. LINDBERG, Waukegan, Ill.

The Women's Auxiliary—Organization, Management, and Special Problems. MRS. G. E. GOODYEAR, Hastings, Mich.

Public Relations—Promoting a Public Relations Program. CHARLOTTE GARRISON, R.N., Battle Creek, Mich.

Hospital Standardization—Meeting the Requirements for Approval by the American College of Surgeons. E. W. WILLIAMSON, M.D., Chicago.

Collaborators: GRAHAM L. DAVIS, Battle Creek, Mich.; PAUL H. FESLER, Nopeming, Minn.

Wednesday, 7:45 a.m.—Private Dining Room, Stevens Hotel

Breakfast Conference, Sponsored by the American Association of Medical Record Librarians in co-operation with the American College of Surgeons. The Medical Records Librarians' Program for Self Development. RAY M. AMBERG, Minneapolis, presiding.

Discussion from the following aspects:

Approved Courses for Medical Records Librarians. ALICE G. KIRKLAND, Oakland, Calif.

Need for Courses for Medical Records Librarians Already in the Field. EDNA K. HUFFMAN, Chicago.

Self Study and Personal Development. MARGUERITE E. HOOVLER, Pittsburgh.

General Discussion. Opened by SISTER M. PATRICIA, Duluth.

Wednesday, 9:30 a.m.—Upper Tower Ballroom, Stevens Hotel

Joint Conference with American Association of Medical Record Librarians. ROBIN C. BUERKI, M.D., Madison, presiding.

Greetings from the American College of Surgeons to the American Association of Medical Record Librarians. MALCOLM T. MACEACHERN, M.D., Chicago.

Personal, Professional, and Other Qualifications of a Successful Medical Records Librarian. DOROTHEA M. TROTTER, R.R.L., Grand Rapids, Mich.

Guiding Principles of the Medical Records Librarian in Her Relations with the Resident Staff. SISTER MARY SERVATIA, R.R.L., St. Louis.

The Adoption and Putting Into Operation of the Standard Medical Nomenclature. EDWIN P. JORDAN, M.D., Chicago.

Surgical Nomenclature. HILGER P. JENKINS, M.D., Chicago.

The Practical Application of the Punch Card System in Assembling Statistical and Research Data from the Medical Records. JESSIE L. HARNED, R.R.L., Durham, N. C.

Diagnosis Code for Use in Tabulating Morbidity Statistics. HALBERT L. DUNN, M.D., Washington; Chief Statistician for Vital Statistics of the United States Bureau of Census.

Open Forum Discussion: Problems Pertaining to the Obtaining of Good Medical Records. Conducted by ROBIN C. BUERKI, M.D., Madison, Wis.

Wednesday, 1:00 p.m.—Lounge, 2nd Floor, Stevens Hotel

Consultation Service: Administrative practices and policies; internships and residencies; physical therapy; obstetrical service in the hospital; legal aspects of the hospital; hospital standardization problems.

Wednesday, 2:30 p.m.—Local Hospitals.

Group Conferences and Demonstrations.

Augustana Hospital. E. I. ERICKSON, Superintendent.

Central Service for Dressings. AGNES HANSON, R.N.

Rehabilitation and Modernization of the X-ray Department. DAVID S. BEILIN, M.D.

Management of the Pharmacy Service. FRANCES DOUGLAS, Ph.G.

Children's Memorial Hospital. MABEL W. BINNER, R.N., Superintendent.

Outpatient Clinic Management; Inspection of New Clinic Building. BABETTE S. JENNINGS.

Organization and Management of Laundry and Linen Service. ALOIS J. ULRICH.

Purchasing and Stores. CARRIE H. JONES.

Cook County Hospital. GENERAL MANUS MCCLOSKEY, Warden.

Central Control of Parenteral Solutions. FREDERICK STEIGMANN, M.D.

Preservation and Use of Medical Records in a Large Hospital. STELLA FORD WALKER.

Organization and Management of a Blood Bank Service. ELIZABETH SCHIRMER, M.D.

Grant Hospital. CLINTON F. SMITH, Administrator.

Central Stores; Perpetual Inventory; Purchasing. ROLAND A. SCOTT.

Central Service. MARGARET KARSTEN MIHULA.

Medical Records Librarians' School. EDNA K. HUFFMAN.

Department of Nursing: Conference Featuring Ward Instruction and Ward Supervision. ELIZABETH C. WIVEL, R.N.

Henrotin Hospital. VERONICA MILLER, R.N., Administrator.

Office Management. HENRIETTA DRAGER.

Central Control of Supplies. MILDRED KRAUSE, R.N.

Housekeeping. MILDRED PAGE.

Michael Reese Hospital. ALBERT H. SCHEIDT, Associate Director.

Personnel Management. GEORGE PECK.

Clinic Management and Medical Social Service. ELIZABETH MCCONNELL and LEONORA RUBINOW.

Modern Laundry Methods. R. J. GABRIELSON.

Presbyterian Hospital. ASA S. BACON, Superintendent.

Personality in the Hospital—the Front Office—Contact and Dealing with Patients, Relatives, and Friends. ASA S. BACON.

Business Methods—Accounting, Purchasing, Income, Charges, Control. HERMAN HENSEL and L. D. REID.

Centralized Food Service. BEULAH HUNZICKER.

St. Joseph Hospital. SISTER ZITA, A.M., Administrator.
Organization and Management of the Obstetrical Department: Rehabilitation of the Physical Plant. CLYDE GEIGER, M.D., and EMILY MARCOULIER.
Operating Room Management, Technique and Procedures; Rehabilitation of the Physical Plant. SISTER DELPHINE, R.N., B.S.
Food Administration. SISTER RUFINA, R.N., B.S.
University of Chicago Clinics. G. OTIS WHITECOTTON, M.D., Superintendent.
Anesthesia; Operating Room Management and Technique. HUBERTA LIVINGSTONE, M.D., and DOROTHY SCHMIDT, R.N.
Food Service. ELLA M. ECK.
Medical Records Library; Creation of a Medical Record. ADALINE HAYDEN.
Women and Children's Hospital. EDNA H. NELSON, Superintendent.
Mother's Milk Bureau. MARION GREENE.
Care of Newborn. MARIE HARLAN.
Obstetrical Technique and Procedures. BEATRICE E. TUCKER, M.D.

Wednesday, 7:00 p.m.—Upper Tower Ballroom, Stevens Hotel

Motion pictures.
Departmental Panel Round Table Conference. Conducted by ROBIN C. BUERKI, M.D., Madison, Wis., and MALCOLM T. MACEACHERN, M.D., Chicago.
Administrative Practices. THOMAS T. MURRAY, Albany.
Anesthesia Service. HUGH A. CUNNINGHAM, M.D., Milwaukee.
Business Management. WILLIS J. GRAY, Detroit.
Food Service. HELEN MAE BRYAN, Chicago.
Laundry and Linen Service. ALOIS J. ULRICH, Chicago.
Medical Records. MARGARET C. TAYLOR, Rochester, N. Y.
Medical Social Service. JOSEPHINE G. TAYLOR, Chicago.
Medical Staff. CHARLES B. PUESTOW, M.D., Chicago.
Nursing Service. ELIZABETH ODELL, Evanston, Ill.
Obstetrical Service. CARL P. HUBER, M.D., Indianapolis.
Outpatient Department. ALMA VAUPEL, Milwaukee.
Pathological Service. LALL G. MONTGOMERY, M.D., Muncie, Ind.
Patients' Libraries. SELMA LINDEM, Chicago.
Physical Therapy. JOHN S. COULTER, M.D., Chicago.
Tuberculosis. M. POLLAK, M.D., Peoria, Ill.
Utilities. OSCAR E. OLSON, Madison, Wis.
X-ray Service. JAMES T. CASE, M.D., Chicago.

Thursday, 7:45 a.m.—Private Dining Room, Stevens Hotel

Breakfast Conference. The Public Relations Program of Your Hospital. ALDEN B. MILLS, Chicago, presiding.
Discussion from the following aspects:
Newspaper Publicity. LAURA JACKSON, Chicago.
Annual Reports. ASA S. BACON, Chicago.
Syndicated Material. ALBERT G. HAHN, Evansville, Ind.
Fund Raising. ADA B. MCCLEERY, R.N., Evanston, Ill.
General Discussion. Opened by CARL I. FLATH, Detroit.

Thursday, 9:30 a.m.—Upper Tower Ballroom, Stevens Hotel

FRANK R. BRADLEY, M.D., St. Louis, presiding.
Hospital Administration and Its Relation to Organized Medicine. BORIS FINGERHOOD, Brooklyn.
The Problem of Tuberculosis in the General Hospital. ERNEST S. MARIETTE, M.D., Oak Terrace, Minn.
An Organized Health Service for Hospital Employees. ROBERT E. NEFF, Iowa City, Iowa.
The Implications of Good Medical Staff Organization. JOSEPH G. NORBY, Milwaukee.
Hospital Rates. ALBERT H. SCHEIDT, Chicago.
General Discussion. Led by CHARLES S. WOODS, M.D., Peoria, Ill.

Thursday, 2:30 p.m.—Local Hospitals

Group Conferences and Demonstrations.
Chicago Lying-In Hospital and Dispensary. S. A. FERGUSON, Assistant to Director.
Registration and Admission. JEAN B. ANDERSON.
Isolation of Obstetrical Patients. GEORGIA HUKILL, R.N.
Nursery Management. GEORGIA HUKILL, R.N.
Mount Sinai Hospital. STEPHEN MANHEIMER, M.D., Director.
Medical Staff Organization and Administrative Control. STEPHEN MANHEIMER, M.D.
Clinical Laboratory—Organization, Management and Service. I. DAVIDSOHN, M.D.
Business Management—Accounting, Purchasing and Revenue. NATHAN W. HELMAN, C.P.A.
Passavant Memorial Hospital. IRVING S. CUTTER, M.D., Superintendent.
Oxygen Therapy. M. HERBERT BARKER, M.D.
Physical Therapy. JOHN S. COULTER, M.D.
Engineering and Maintenance. WILLIAM W. DAVISON.
Ravenswood Hospital. J. DEWEY LUTES, Administrator.
General Administration of a 200 Bed Hospital. J. DEWEY LUTES.
Medical Records Department. HELENA BENDER.
Medical Library. LEONORA PERRY.
Research and Educational Hospitals. MAJOR H. WORTHINGTON, M.D., Managing Officer.
Psychiatry in a General Hospital; Inspection of New Neuro-Psychiatric Building. I. R. SONENTHAL, M.D.
Central Supply Service and Central Linen Exchange. ANN LUCILLE LAIRD.
Study Tour of Orthopedic Hospital. ANTOINETTE J. RADEK, R.N., Assistant Chief Nurse.
St. Elizabeth Hospital. SISTER M. ADOLPHINE, R.N., Superior.
Obstetrical Department in a General Hospital. J. R. LAVIERI, M.D.
Central Supply Service. SISTER M. MARGARITIS, R.N.
Modernized Nursery Technique. MARIE DALTON, R.N.
St. Luke's Hospital. CHARLES A. WORDELL, Director.
Business Methods—Accounting, Purchasing, Stores, Preparation of Budget, Monthly Reports. FREDERICK T. MUNCIE.
Equipment and Procedures for Dressing Cart, Modified Wangensteen or Gravity Suction. EVELYN VAN DE STEEG.
Set-Up in Major Operating Rooms with Special Reference to Personnel; Blood Transfusions. MARIE ANTE.
Wesley Memorial Hospital. E. R. SNYDER, Superintendent.
Practical Problems in Anesthesia and Oxygen Therapy. MARY KARP, M.D.
Food Service. ELIZABETH H. TUFT.
Obstetrical Procedures. VERTE J. JOHNSON.
West Suburban Hospital. L. C. VON DER HEIDT, Superintendent.
Management of the Newborn; Humidity a Dominant Nursery Safeguard. LEONORA SCHULZE, R.N., MARY MOLLEY, R.N., and L. C. VON DER HEIDT.
Palmprint Identification. GILBERT P. POND, M.D.
Maintenance of the Physical Plant and Engineering. DAVID PATTERSON.
Woodlawn Hospital. GRACE L. DEVILBISS, R.N., Superintendent.
A Study of Organization, Management and Procedures in a Private Hospital of 100 Beds. GRACE L. DEVILBISS, R.N., and Staff.

PRELIMINARY CLINICAL PROGRAM

ARRANGED IN THE FOLLOWING SUBDIVISIONS: GENERAL SURGERY, OBSTETRICS AND GYNECOLOGY, GENITO-URINARY SURGERY, FRACTURES AND OTHER TRAUMAS, ORTHOPEDIC SURGERY, THORACIC SURGERY, NEUROSURGERY, OPHTHALMOLOGY, OTOLARYNGOLOGY

GENERAL SURGERY

Monday

CHICAGO MEMORIAL HOSPITAL

PETER S. CLARK, CHARLES J. DRUECK, ROBERT A. MELENDY, BENNETT R. PARKER, JOHN VAN PROHASKA, M. L. WEINSTEIN, LEO M. ZIMMERMAN, and GEORGE M. LANDAU—1. Operative and dry clinic:

JOHN VAN PROHASKA. Surgical aspects of carcinoma of the ampulla of Vater; enterogenous cysts in infants.

M. L. WEINSTEIN. Resection of stomach with DePetz instrument; rectal anesthesia.

LEO M. ZIMMERMAN. Essential problems in the surgical treatment of inguinal hernia.

BENNETT R. PARKER. Problems in gall-bladder surgery.

COOK COUNTY HOSPITAL

Staff—2. Symposium on biliary tract disease:

EDMOND F. FOLEY. Medical aspects.

HANS L. POPPER. Perverted physiology.

FREDERICK STEIGMAN. Jaundice.

HARRY CULVER. Urological factors in differential diagnosis.

WALTER SCHILLER. Pathology.

M. J. HUBENY. Roentgenological aspects in diagnosis.

LINDON SEED. Clinical results with synthetic Vitamin K in jaundice.

RAYMOND W. MCNEALY. Pre- and postoperative management.

KARL A. MEYER. Operative management.

MARSHALL DAVISON. Post-cholecystectomy pain.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

MICHAEL REESE HOSPITAL

Staff—2. Symposium on transfusion and infusion:

LEO M. ZIMMERMAN. History of blood transfusion.

SIDNEY O. LEVINSON. General survey of a modern transfusion department.

HAROLD LAUFMAN. Blood transfusion reactions, statistical study.

ANNA MARIE STRAUSS. Experiences with indirect transfusion reactions.

HEINRICH NECHELES. Plasma and serum as substitutes for whole blood transfusions; physiology and laboratory experiments.

FRANK E. RUBOVITS. Clinical experience with plasma and serum transfusions.

Inspection of transfusion and serum departments.

RESEARCH AND EDUCATIONAL HOSPITALS

Scientific Exhibit: Cancer of the large bowel and appendicitis.

Tuesday

AUGUSTANA HOSPITAL

NELSON M. PERCY and OSCAR E. NADEAU—9. Operative and dry clinics.

ALBERT MERRITT BILLINGS HOSPITAL

L. R. DRAGSTEDT—9. Surgery of the colon.

HILGER P. JENKINS—9. Plastic surgery of the face, cases and operations.

COOK COUNTY HOSPITAL

KARL A. MEYER—9. Operative clinic: Stomach and colon.

JOHN R. HARGER—9. Operative clinic: Peridural anesthesia for major operations.

C. C. GUY—9. Surgical problems in the diabetic.

SUMNER L. KOCH—9. Dry clinic: Injuries and infections of the hand.

RAYMOND W. MCNEALY—10. Operative clinic: Gall-bladder; gastric surgery.

E. M. MILLER—10. Dry clinic: Bowel obstruction in the newborn; gangrene complicating scarlet fever; volvulus of sigmoid colon in children.

J. D. KOUCKY—10. Operative and dry clinic: Arteriosclerotic gangrene of the legs.

JOHN B. O'DONOGHUE—10. Operative clinic: Gastrointestinal.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

Staff—1. Demonstration on cadaver, some principles in gall-bladder surgery.

EVANGELICAL HOSPITAL

P. E. HOPKINS, G. E. JOHNSON, and JAMES PATEJDL—9. Operations.

EVANSTON HOSPITAL

W. R. PARKES, FREDERICK CHRISTOPHER, W. KENNETH JENNINGS, J. PEERMAN NESSELROD, JAMES P. GRIER, J. E. KEARNS, and FREDERICK WILLIAM MERRIFIELD—9. Operative and dry clinic:

FREDERICK CHRISTOPHER. Abdomino-perineal resection for carcinoma of the rectum.

W. KENNETH JENNINGS. The colostomy.

JAMES P. GRIER. Surgery of the biliary tract.
J. E. KEARNS. Vascular clinic.
J. PEERMAN NESSELROD. Motion pictures of the rectum.
FREDERICK W. MERRIFIELD. Oral surgery.

GRANT HOSPITAL

KARL A. MEYER—9. Operations.
A. G. ZIMMERMAN—9. Mediastinal tumors.

MICHAEL REESE HOSPITAL

Staff—9. Operative clinic:
JAMES PATEJDL. Gastro-intestinal.
R. B. BETTMAN and WILLIAM TANNENBAUM. Gall-bladder.
A. A. STRAUSS and S. F. STRAUSS. Stomach and colon.
S. L. GOLDBERG. General surgery.
Staff—9. Dry clinic: Symposium on peptic ulcer.
HEINRICH NECHELES. Newer conceptions of the physiology of stomach in relation to ulcer.
JACOB MEYER. The bleeding ulcer.
H. F. BINSWANGER. Treatment of bleeding ulcer with colloidal preparations.
S. F. STRAUSS. Indications for gastric surgery.
A. A. STRAUSS. Subtotal gastric resection.
JAMES PATEJDL. New methods in gastric surgery.
Staff—2. Dry clinic:
LEO M. ZIMMERMAN. Anatomy of inguinal hernia in relation to surgery.
NATHAN N. CROHN. Incidence of strangulation in inguinal hernia; treatment of hernia.
LEO M. ZIMMERMAN and HAROLD LAUFMAN. Unusual complications of inguinal hernia.
R. B. BETTMAN and WILLIAM TANNENBAUM. Wound evisceration.
S. L. GOLDBERG. Septicemia.
D. C. STRAUSS. Epiphyseal separations.
Staff—2. Dry clinic: Peripheral vascular surgery.
LEO M. ZIMMERMAN and S. MUSCOVITZ. Thrombophlebitis and pulmonary embolism.
H. A. ROTH. Indications and technique of saphenous vein ligation.
SAMUEL PERLOW and S. S. HALPERN. Prostigmin in peripheral vascular disease.
SAMUEL PERLOW. Injection treatment of angina pectoris; sympathectomy for peripheral vascular disturbances.
S. STAGMAN and E. BLUMENTHAL. Treatment of varicose veins in the aged.
SAMUEL PERLOW and E. BLUMENTHAL. Ligation of the lesser saphenous vein.

MUNICIPAL TUBERCULOSIS SANITARIUM

CLEMENT L. MARTIN—9. Anorectal tuberculosis.

RAVENSWOOD HOSPITAL

Staff—9. Operations.
Staff—10. Tumor clinic: Scientific exhibit; demonstration of procedure.
Staff—10. End-results of varicose vein operations.

RESEARCH AND EDUCATIONAL HOSPITALS

GÉZA DE TAKATS, J. T. REYNOLDS and staff—9. Operative clinic and symposium on hypertension: Splanchnic nerve section for hypertension.
S. R. ROSENTHAL. Renal biopsies.
F. K. HICK. Physiology of the heart in hypertension.
H. C. LUETH. The kidney function in hypertensive states.
H. E. HEYER. The brain in hypertensive states.
R. O. RISER. The eye grounds in hypertension—illustrated by kodachrome slides.
GÉZA DE TAKATS and associates. The surgical approach to the problem of hypertension.

Scientific Exhibit

Cancer of the large bowel and appendicitis.

ST. JOSEPH HOSPITAL

Staff—9. Operative and dry clinics:
HUGH MCKENNA. Cholecystectomy; thyroidectomy; closure of colon following second-stage miculiz operation for carcinoma of descending colon.
WILLIAM BECK. Acute intestinal obstruction.
B. J. FITZGERALD. Thyroidectomy.
LEONARD KRATZ. Ruptured appendicitis.
FRANK MCCARTY. Thyroidectomy; cholecystectomy; stomach surgery; gall-bladder disease.
J. A. WIMBERLY. Thyroidectomy.
STEPHEN A. ZEIMAN. Herniotomy; moving pictures of hernia operation.

ST. LUKE'S HOSPITAL

W. R. CUBBINS—9. Operations.
SELIM W. MCARTHUR—9. Operations.
Staff—9. Dry clinic:
E. C. HOLMBLAD and R. A. JACOBSON. Treatment of gas gangrene.
T. L. HANSEN and J. M. L. JENSEN. Shelf fractures of tibia.
JOHN D. ELLIS. Amputations.
JOHN L. LINDQUIST. Physiological problems and management in postoperative gastro-intestinal constant suction drainage.
G. V. PONTIUS. Hypertrophic pyloric stenosis in the adult.
H. I. MEYER. Paget's disease; osteitis deformans.
W. R. CUBBINS. Combined abdominal and chest injuries with differential diagnosis.
ERIC OLDBERG. Spinal injuries.
ALFRED P. SOLOMON. Personality disorders during retarded convalescence following industrial injuries.
H. E. MOCK and associates—9. Skull fracture exhibit.
PAUL W. GREELEY—10. Operations: Total reconstruction of external ear; rhinoplasty.

WESLEY MEMORIAL HOSPITAL

RAYMOND W. MCNEALY—9. Thyroidectomy; resection of colon.
S. J. FOGELSON—9. Gastric resection.
WILLIAM M. MCMILLAN—9. Repair of inguinal hernia.
JOSEPH E. SCHAEFER and KENNETH W. PENHALE—9. Repair of cleft palate.
WILLIAM A. HENDRICKS—9. Demonstrations: Lymphosarcoma of the stomach; stab wound of the heart with repair; isolated simple non-specific ulcer of the cecum, healing without surgery.
GUY S. VAN ALSTYNE—9. Demonstration: Common duct stone with rupture of common duct.
EARL O. LATIMER—10. Gastric resection.
WILLIAM MILLER—10. Cholecystectomy.
NORMAN G. PARRY—10. Cholecystectomy.

Wednesday

AUGUSTANA HOSPITAL

A. T. LUNDGREN and EARL GARSIDE—9. Operative and dry clinic.
JOHN W. NUZUM and RUDOLPH ODEN—9. Operative and dry clinic.

CHICAGO MEMORIAL HOSPITAL

Medical, surgical x-ray and pathological departments—2. Symposia: Treatment of pneumonia with serum and sulfapyridine; peptic ulcer.

CHILDREN'S MEMORIAL HOSPITAL

ALBERT H. MONTGOMERY, JOHN A. GRAHAM, JAY IRELAND, W. J. POTTS, J. J. MUSSIL, A. E. DIGGS, and S. E. LAWTON—9. Operations.

COOK COUNTY HOSPITAL

VICTOR L. SCHRAGER—9. Operations.
RALPH C. SULLIVAN—9. Operations.
EDWARD A. CHRISTOFFERSON—9. Operative clinic: Appendicitis.
FRANK J. JIRKA—10. Dry clinic: Dysgerminoma in the male.
J. R. BUCHBINDER—1. Operative clinic: Thyroid; biliary tract.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

Staff—1. Demonstrations on cadaver and anesthetized dog: Gastric surgery.

HENROTIN HOSPITAL

ROBERT F. SHARER—9. Operative and dry clinic: Gastrojejunal ulcers.

JACKSON PARK HOSPITAL

C. C. CLARK—9. Abdominal operations.
A. BAMBERGER—10. Abdominal and thyroid operations
H. H. COX—10. Operative and dry clinic.

LORETTO HOSPITAL

E. PERRY VAUGHN—9. Operations.
ALLEN E. STEWART and L. B. NEWMAN—10. Operative and dry clinic.
FRANK M. SYLVESTER—10. Operations.
LOUIS F. PLZAK and FRANK F. KOTALIK—10. Goiter clinic.

MERCY HOSPITAL

M. F. MCGUIRE—9. Operative and dry clinic: Biliary tract.
WILLIAM J. PICKETT—9. Operative and dry clinic: Gastric surgery.
C. F. SAWYER—9. Intestinal obstruction.
J. SANDROY—9. Tests for liver function.

MICHAEL REESE HOSPITAL

Staff—9. Operative clinic:
M. L. PARKER. Thyroid.
A. A. STRAUSS and S. F. STRAUSS. Gastro-intestinal.
R. B. BETTMAN and WILLIAM TANNENBAUM. Surgery of extra hepatic bile tracts.
NATHAN N. CROHN. Subject to be announced.
LEO M. ZIMMERMAN. Hernia.

Staff—9. Dry clinic: Symposium on diseases of the bile tracts:
HEINRICH NECHELES. New knowledge of physiology of the gall-bladder.
S. A. PORTIS. Medical indications for surgery of the gall-bladder.
R. B. BETTMAN. Technique of gall-bladder operations with motion pictures.
A. M. SERBY. Liver function tests.
WILLIAM TANNENBAUM. Carcinoma of the gall-bladder.
G. M. LICHTENSTEIN. End-results of gall-bladder surgery.
NATHAN N. CROHN. Measurements of the common and cystic ducts.
ROBERT ARENS and R. B. BETTMAN. Immediate cholangiography.

Staff—2. Dry clinic:
LEON BLOCH. Ulcerative colitis.
A. A. STRAUSS. Indications for ileostomy in colon surgery.
S. F. STRAUSS. Surgical lesions of the colon.
ROBERT ARENS. X-ray diagnosis of diseases of the colon.
OTTO SAPHIR. Pathology of the colon.
HEINRICH NECHELES. Newer physiology of the intestines.

MOTHER CABRINI MEMORIAL HOSPITAL

E. J. CHESROW and associates—9. Abdominal operations.

PASSAVANT MEMORIAL HOSPITAL

SUMNER L. KOCH, MICHAEL L. MASON, and HARVEY ALLEN—9. Dry clinic: Presentation of cases illustrating problems in surgery of the hand; discussion of wound infections.

RESEARCH AND EDUCATIONAL HOSPITALS

WARREN H. COLE and staff—9. Operative clinic and thyroid symposium: Thyroidectomy; prerequisites for thyroidectomy.
R. B. MALCOLM. Surgical pathology of goiter.
J. T. REYNOLDS. Postoperative treatment.
CHARLES B. PUESTOW. Suture material and end-results.
LINDON SEED. King operation for recurrent nerve paralysis.
R. W. KEETON. Management of patients with hyperthyroidism and cardiac disease.
P. W. GREELEY—9. Thick razor graft to axillary contracture; island artery flap to nose; costal cartilage graft to nose; full thickness skin graft to hand; demonstration of plastic cases.
P. J. SARMA—9:30. Varicose vein clinic.
WARREN H. COLE—11:15. Splenectomy.

Scientific Exhibit

Cancer of the large bowel and appendicitis.

ST. ANNE'S HOSPITAL

G. F. THOMPSON, J. J. GEARIN, J. L. KNAPP, J. W. KEANE ADOLPH KRAFT, and H. M. PETERSON—9. Operations.
ADOLPH KRAFT—1. Dry clinic: Carcinoma of cecum.
G. F. THOMPSON—1. Dry clinic: Carcinoma of breast; blastomycosis of cecum.

ST. BERNARD'S HOSPITAL

WILLIAM G. EPSTEIN—9. Thyroid operations.
L. B. DONKLE, G. M. CUSHING, and WILLIAM MULHOLLAND—9. Abdominal operations.
S. L. GOVERNALE—9. Dry clinic: Pseudohypertrophic muscular dystrophy.

ST. LUKE'S HOSPITAL

H. E. MOCK—9. Operations.

Staff—9. Dry clinic:
SELIM W. MCARTHUR. Stomach deformities simulating malignancy in x-ray study.
F. L. MCMILLAN. Lesions of the colon.
H. E. JONES. Treatment of complete biliary fistulas.
E. LEE STROHL. Common duct obstructions.

W. G. DIFFENBAUGH. Autoplastic operation for hernia repair; statistical report.
H. E. MOCK. Breast tumors; malignancies and granulomas of the gastro-intestinal tract.
E. W. RYERSON. Conservative treatment of fractures.
T. P. GRAUER. Injuries of the genito-urinary tract.
HAROLD A. SOFIELD. Fractures of femoral neck.
H. E. MOCK and associates—9. Skull fracture exhibit.

SWEDISH COVENANT HOSPITAL

R. F. ELMER, W. B. STROMBERG, and A. C. PETERSON—9. Operative clinic: Surgery of stomach and gall tracts.
K. L. VEHE, A. P. SANDAHL, and R. E. TALBOTT—9. Head and neck infections; anatomical demonstrations.
M. R. BROMAN—9. Problems in pathology in a private hospital.
R. G. WILLY—9. Diagnostic problems studied in a private hospital.

VETERANS ADMINISTRATION FACILITY

P. F. BROWN and B. F. WARD—9. Abdominal operations.
R. B. MORELAND—9. Operative clinic: Tumors.
A. E. WILLIAMS—9. Inspection tour of deep x-ray and radium unit.
G. R. ALLABEN—10. Dry clinic: Tumor clinic in the general hospital; method of presentation of cases before Tumor Board; diagnosis, treatment, and follow-up.
MAX CUTLER—11. Dry clinic: Carcinoma of the mouth and larynx; problems in diagnosis and treatment; indications for surgery and irradiation in laryngeal carcinoma; presentation of cases.

Thursday

ALEXIAN BROTHERS' HOSPITAL

WILLIAM J. SWIFT—9. Herniotomy.
DANIEL E. MURPHY—9. Circulatory disturbances of lower extremity.
FREDERICK A. RETTIG—10. Abdominal surgery and post-operative care.

AUGUSTANA HOSPITAL

NELSON M. PERCY and OSCAR E. NADEAU—9. Operative and dry clinics. Symposium on diagnosis and surgery of goiter.

CHICAGO MEMORIAL HOSPITAL

CASPER M. EPSTEIN—9. Operative clinic: Correction of cleft palate; facial plastic.

CHILDREN'S MEMORIAL HOSPITAL

ALBERT H. MONTGOMERY, JAY IRELAND, and J. J. MUSSIL—2:30. Demonstration of cases: Abdominal tumors in children; subphrenic abscess, osteomyelitis, retroperitoneal tumors.

COLUMBUS HOSPITAL

DANIEL A. ORTH—9. Operations.
H. E. DAVIS—9. Selective radiation therapy of malignancies; presentation of cases.
R. J. MURPHY—9. Operations.
ERNEST NORA—9. Demonstration: Stethocardiograph as an aid in surgical diseases.
H. R. KENNY—10. Crohn's disease; presentation of cases.

COOK COUNTY HOSPITAL

RALPH C. SULLIVAN—9. Operative clinic.
MARSHALL DAVISON—10. Operative clinic: Thyroid.
R. B. BETTMAN—2. Operative clinic.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

RAYMOND W. MCNEALY—11. Lecture: Femoral hernia.
Staff—1. Demonstration on cadaver: Surgery of the large bowel.

EVANGELICAL HOSPITAL

P. E. HOPKINS, G. E. JOHNSON, and JAMES PATEJDL—9. Operations.

HOLY CROSS HOSPITAL

V. F. TORCZYNSKI. Blood pressure and spinal anesthesia—study of 100 cases.
J. B. KARR. Injection treatment of hernia; herniotomy.
F. F. FRAIDER and N. B. PAVLETIC. Indirect inguinal hernia.
F. J. SALETTA. Thyroidectomy.
M. J. BADZMIEROWSKI. Thyroidectomy.
J. F. RUZIC and D. S. DICIRO. Hernioplasty; vaginal hysterectomies; cholecystectomy; common duct exploration; thyroidectomy.

ILLINOIS MASONIC HOSPITAL

WILBUR E. KEESEY and J. WALTER JOHNSON—9. Cholecystectomy.
R. BRUCE MALCOLM—9. Partial gastrectomy.
JOHN R. HARGER and JOHN PISHOTTA—9. Cholecyst-duodenostomy.
C. DRUECK, SR. and HENRY E. OLIVER—9. Hemorrhoidectomy.
W. C. BORNEMEIER and I. KROSS—10. Herniotomy.
T. G. WALLIN and BEULAH WALLIN—10. Amputation of breast.
WARREN PUGH—10. Resection of colon.
JOHN F. DAVIS and A. E. WUESTEMAN—11. Colostomy.
PAUL MORF and EDWARD LEVISOHN—11. Appendectomy.
W. B. GERHARD and GEORGE GEYMER—11. Amputation of breast.

MERCY HOSPITAL

L. D. MOORHEAD—9. Operative and dry clinic: Thyroid.
A. K. VAUGHN—9. Treatment of varicose veins and ulcers.

MICHAEL REESE HOSPITAL

Staff—9. Operative clinic:
A. A. STRAUSS and S. F. STRAUSS. Gastro-intestinal.
M. L. PARKER. Subject to be announced.
JAMES PATEJDL. Stomach and colon.
S. L. GOLDBERG. Subject to be announced.
SAMUEL PERLOW. Peripheral vascular surgery.
Staff—9. Dry clinic: Thyroid symposium.
S. SOSKIN. Physiological disturbances in thyrotoxicosis.
ROBERT C. LEVY. Special tests in diagnosis of borderline thyroid cases.
OTTO SAPHIR and LEO M. ZIMMERMAN. Neoplasms of the thyroid.
M. L. PARKER and H. A. ROTH. Analysis of five years' thyroid surgery and follow-up.
A. K. KOFF. Menstrual disturbances in relation to thyroid diseases.
Staff—2. Dry clinic:
S. L. GOLDBERG. Fish bone perforation of ileum.
M. L. PARKER. Periarteritis nodosa of bowel; foreign bodies in the stomach; regional ileitis.

R. B. Bettman and William Tannenbaum. Foreign body perforation of the intestinal tract; complications of gastro-enterostomy.
S. L. Goldberg and L. N. Katz. Attempts at reducing experimental hypertension in dogs.

MUNICIPAL TUBERCULOSIS SANITARIUM

R. M. Davison—9. Surgical treatment of tuberculosis.

NORWEGIAN-AMERICAN HOSPITAL

J. M. Amberson—9. Intravenous medication.
G. B. Fauley—9:30. Surgical physiology of the thyroid.
A. M. Jensen—9:30. Sulfanilamide in general surgery.
J. R. Orndorff—10. Technique of thyroidectomy.
F. H. Fowler—10. Time question in appendicitis.
J. V. Fowler, Jr.—10:30. Pre- and postoperative care of the thyroid patient.
J. E. Verhaag—10:30. Hernia from industrial causes.
M. M. Corbett—10:30. Immediate treatment of abdominal injuries.
Warren Johnson—11. Carcinoma of the stomach.
F. M. Nicholson—11. Treatment of scalp injuries.
M. E. Lichtenstein—11:30. Technique of cholecystectomy.
J. V. Fowler, Sr.—11:30. Carcinoma of the breast.

PASSAVANT MEMORIAL HOSPITAL

John A. Wolfer and associates—9. Tumor clinic:
Frank Queen. The surgeon and pathologist as a diagnostic team, i.e., the decision for and conduct of biopsy with illustrative cases.
Earl E. Barth. Important preliminaries prior to x-ray therapy.
H. E. Davis. Selection of tumor cases and x-ray vs. radium therapy.
H. L. Jaffe. Administration of radiation therapy for malignant tumors.
F. W. Merrifield. Our results with intra-oral tumors.
J. M. Greene. Useful aids in differential diagnosis of tumors of the neck.
John A. Wolfer. Our results with cancer of the breast.
E. M. Smith, Jr. Results with treatment of unusual cutaneous tumors.
Herman Chor. The psychiatrist's part in a tumor clinic; illustrative cases.
Earl E. Barth. Inspection of radiation therapy department of Northwestern University Medical School.

PRESBYTERIAN HOSPITAL

Kellogg Speed, Carl B. Davis, Albert H. Montgomery, Edwin M. Miller, Francis Strauss, Harry Oberhelman, W. J. Potts, Hillier Baker, Frank V. Theis, Egbert H. Fell, R. K. Gilchrist, and Vernon C. David—9. Operations.

RESEARCH AND EDUCATIONAL HOSPITALS

Charles B. Puestow and staff—9. Operative and dry clinic:
Charles B. Puestow. Choledochostomy for stone; physiology of the common duct as related to surgery.
E. F. Foley. The jaundiced patient from the medical viewpoint.
Warren H. Cole. Lesions of the cystic duct as related to gall-bladder disease.
Lindon Seed and J. Karabin. Use of Vitamin K in hypoprothrombinemia.
Charles B. Puestow. Excision carcinoma of colon.

Scientific Exhibit

Cancer of the large bowel and appendicitis.

ST. LUKE'S HOSPITAL

Géza de Takats and J. T. Reynolds—9. Operative clinic: Lumbar sympathectomy.
Paul W. Greeley—9. Operations: Construction of new eyebrows; full thickness skin graft; split skin graft.
H. E. Mock and associates—9. Skull fracture exhibit.
John S. Coulter—10. Physical therapy in peripheral vascular disease.
G. K. Fenn—10. Extracardiac factors influencing coronary circulation.
G. W. Scupham—10. Renal disease and hypertension.
R. B. Capps—11. Vascular reflexes during operations.
J. M. L. Jensen—11. Paravertebral block of the sympathetics; technique and indications.
Géza de Takats—11. Demonstration in vascular surgery: Cervical rib and aneurysm; wired aneurysm of aorta; congenital arteriovenous fistula of the hand; modified Kondoleon operation of thrombophlebitic induration.

ST. MARY OF NAZARETH HOSPITAL

T. M. Larkowski—9. Operative clinic.
E. H. Warszewski—10. Operative clinic.
Theodore Steinert—10. Operative clinic.
G. M. Mueller—11. Operative clinic.

Friday

ALBERT MERRITT BILLINGS HOSPITAL

Alexander Brunschwig—9. Operative clinic: Tumors of the adrenal gland.

CHILDREN'S MEMORIAL HOSPITAL

Louis W. Schultz—1. Operative clinic and demonstration of cases: Oral surgery.

COOK COUNTY HOSPITAL

Louis River—9. Dry clinic: Abdominal incisions.
E. H. Warszewski—10. Operative clinic: Reconstruction treatment of burns.
J. R. Buchbinder—1. Operative clinic: Gastric resection for duodenal ulcer.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

MICHAEL REESE HOSPITAL

James Patejdl, A. A. Strauss, S. F. Strauss, M. L. Parker, Nathan N. Crohn, S. L. Goldberg, Leo M. Zimmerman, and Samuel Perlow—9. Operations.

MT. SINAI HOSPITAL

D. A. Willis—9. New type of hernioplasty.
E. I. Greene—9. Operative clinic.
V. L. Schrager and staff—9. Hernioplasty.
A. A. Strauss and S. F. Strauss—9. Surgery of the colon.
M. R. Guttman—9. Plastic surgery of the nose.
Jacob Lifschutz—9. Endoscopy.
Emil Aison—9. Oral surgery.
J. M. Mora—9. Thyroidectomy.

PASSAVANT MEMORIAL HOSPITAL

J. R. Buchbinder—9. Dry clinic: Illustrations of end-results in treatment of gastric and duodenal lesions.

PRESBYTERIAN HOSPITAL

FREDERICK B. MOOREHEAD—9. Plastic surgery.

RESEARCH AND EDUCATIONAL HOSPITALS

R. B. MALCOLM and staff—9. Operative and dry clinic:
R. B. MALCOLM. Radical resection for carcinoma of breast; surgical pathology of breast tumors; excision of branchial cleft cyst.
P. J. SARMA. Differential diagnosis of breast lesions.
T. J. WACHOWSKI. Radiation therapy of carcinoma of breast.
GEORGE DE TARNOWSKY. End-results in carcinoma of breast.
P. W. GREELEY—10. Plastic surgery, follow-up clinic.

Scientific Exhibit

Cancer of the large bowel and appendicitis.

ST. ELIZABETH'S HOSPITAL

Staff—9. Operative and dry clinics:
M. G. LUKEN. Hernia operations.
A. G. ZIMMERMAN. Thyroidectomy; treatise on pathology of the thyroid gland.
E. B. KALVELAGE. Surgery of the stomach.
Staff—1. Operative and dry clinics:
J. B. O'DONOGHUE. Complications in surgery of the gall-bladder and ducts.
J. P. WOITALEWICZ. Intestinal surgery.

ST. LUKE'S HOSPITAL

H. E. MOCK and associates—9. Skull fracture exhibit.

Days to be Announced

HOSPITAL OF ST. ANTHONY DE PADUA

F. B. OLENTINE and M. J. BADZMIEROWSKI. Thyroid operations.
J. J. SPRAFKA. Abdominal operations.
S. E. DONLON, H. P. SULLIVAN, FRED EHRMANN, W. H. BRADLEY, JOSEPH ZABOKRTSKY, and R. C. DRURY. Operative and dry clinlc.
FRANK J. JIRKA. Lecture: Convulsions under anesthesia.

OBSTETRICS AND GYNECOLOGY

Monday

COOK COUNTY HOSPITAL

A. J. KOBAK and H. H. HILL—2. Studies in acriflavine and glycerine.
JAMES H. BLOOMFIELD—2. Demonstration of cases: Cesarean section and local anesthesia; management of occiput posterior.
S. J. BENENSOHN—2. Lues in the prenatal clinic.
T. J. MORRIS—2. Puerperal infection.

RESEARCH AND EDUCATIONAL HOSPITALS

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

Tuesday

CHICAGO LYING-IN HOSPITAL

FRED L. ADAIR, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, H. CLOSE HESSELTINE, and FRANKLIN F. SNYDER—9. Operations.
FRED L. ADAIR and staff—2. Dry clinic:
FRED L. ADAIR. Subject to be announced.
FREDERICK W. SCHULTZ. Management of birth trauma.
WILLIAM J. DIECKMANN. Treatment of the toxemias of pregnancy.
M. EDWARD DAVIS. Treatment of placenta previa.
GORDON T. BURNS. Treatment of abruptio placentae.
EDITH L. POTTER. A review of the post-mortem findings in 2,000 fetal and neonatal deaths.
CHARLOTTE L. CLANCY. Problems in the control of reproduction.

COOK COUNTY HOSPITAL

W. T. CARLISLE—9. Gynecological operations.
A. E. KANTER—10. Gynecological operations.

EVANSTON HOSPITAL

W. C. DANFORTH, R. M. GRIER, HOWARD J. HOLLOWAY, P. H. SMITH, and C. E. GALLOWAY—9. Operative clinic: Vaginal hysterectomy; abdominal hysterectomy.

MERCY HOSPITAL

HERBERT E. SCHMITZ and staff—9. Operative and dry clinic: Vaginal hysterectomy; Watkins transposition operation; Manchester-Fothergill operation for vaginal plastic; anterior colporrhaphy and perineorrhaphy; Le Fort colpocleisis.
Staff—2. Demonstrations and discussions of obstetrical problems.

MICHAEL REESE HOSPITAL

J. L. BAER—9. Operation: Radical hysterectomy.
J. E. LACKNER—9. Operation: Total abdominal hysterectomy.
L. E. FRANKENTHAL—9. Operation: Cesarean section.

PASSAVANT MEMORIAL HOSPITAL

ARTHUR H. CURTIS and GEORGE H. GARDNER—9. Operative and dry clinic: Integration of newer anatomical studies with clinical gynecology.

RAVENSWOOD HOSPITAL

Staff—10. Demonstration of cases.

RESEARCH AND EDUCATIONAL HOSPITALS

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

ST. JOSEPH HOSPITAL

CLYDE J. GEIGER—9. Operation for carcinoma of the cervix.

ST. MARY OF NAZARETH HOSPITAL

M. E. UZNANSKI—10. Operations.
A. S. SAMPOLINSKI—11. Vaginal hysterectomy.

WESLEY MEMORIAL HOSPITAL

MARK T. GOLDSTINE and associates—9. Operative and dry clinic: Gynecological.
WILLIAM B. SERBIN—9. Operative and dry clinic: Obstetrical.

GARWOOD C. RICHARDSON—10. Operative and dry clinic: Obstetrical.

Wednesday

CHICAGO LYING-IN HOSPITAL

FRED L. ADAIR, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, H. CLOSE HESSELTINE, and FRANKLIN F. SNYDER—9. Operations.

FRED L. ADAIR and staff—2. Dry clinic.

H. CLOSE HESSELTINE. The use of sulfanilamide, sulfapyridine and sulfathiazole in obstetrics and gynecology.

ROBERT G. BLOCH. Tuberculosis in pregnancy.

FRANKLIN F. SNYDER. Treatment of asphyxia neonatorum.

HENRY T. RICKETTS. Diabetes in pregnancy.

SIMON L. WOLTERS. Treatment of carcinoma of the uterus.

JOHN H. MORTON. Clinical studies in uterine motility.

RUTH WATTS. Studies of ovarian tumors.

CHICAGO MEMORIAL HOSPITAL

HARRY B. W. BENARON, JAMES E. FITZGERALD, WILLIAM F. HEWITT, GEORGE N. SCHIFF, BEATRICE E. TUCKER, and HARRY L. MEYERS—9. Operative and dry clinic:

HARRY B. W. BENARON. Presacral block in obstetrics.

GEORGE N. SCHIFF. Obstetrical analgesia.

JAMES E. FITZGERALD. Effect of Vitamin K in labor.

BEATRICE E. TUCKER and HARRY L. MEYERS. Manchester anterior colporrhaphy and perineorrhaphy.

COOK COUNTY HOSPITAL

HERBERT E. SCHMITZ—9. Gynecological operations.

A. F. LASH—10. Gynecological operations.

EVANGELICAL HOSPITAL

P. ARTHUR DELANEY and associates. Clinical pathological conference on unusual uterine pathology: Endometrial sarcoma; malignancy (leiomyosarcoma) in uterine fibroids; sarcoma botryoides, child 2½ years old; adenomyoma uteri (Frankel), endometriosis; actinomycosis in ovaries; granulosa cell tumor of ovary (microscopic); carcinoma in cystic ovaries; carcinoma in solid ovaries; ectopic tubal pregnancy with tuberculous salpingitis.

LORETTO HOSPITAL

HARVEY LITTLE and NICHOLAS BALSAMO—9. Operative clinic: Gynecological.

MICHAEL REESE HOSPITAL

Staff—9. Dry clinic:

A. F. LASH. Early treatment of puerperal sepsis peritonitis.

H. A. STRAUSS and S. D. MESIROW. Ball method of cephalopelvimetry.

I. F. STEIN. Gynecography.

M. R. COHEN. Clinical use of pregneninolone.

FRANK RUBOVITS. Serum therapy in shock or hemorrhage.

E. J. DECOSTA. Further development of the photostethoscope.

J. E. LACKNER and A. S. TULSKY. Effect of prephysin on the ovary.

PRESBYTERIAN HOSPITAL

N. SPROAT HEANEY, AARON KANTER, EDWARD C. ALLEN, A. H. KLAWANS, FRED PRIEST, and HARRY BOYSEN—9. Operations: Gynecological.

RESEARCH AND EDUCATIONAL HOSPITALS

F. H. FALLS and staff—2. Operative and dry clinic:

W. H. BROWNE. Low cervical cesarean section.

J. R. WOLFF. Six cases of early carcinoma of cervix uteri.

R. A. LIFVENDAHL. Vaginal hysterectomy for prolapse uterus.

F. H. FALLS. Vaginal hysterectomy for fibroids.

H. H. HILL. Report of two cases of ovarian pregnancy.

A. F. LASH. Supracervical hysterectomy.

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

ST. ANNE'S HOSPITAL

J. L. FLEMING—1. Dry clinic: Hematogenous streptococcus puerperal infection; chorion-epithelioma of fallopian tube; inversion of uterus.

R. J. HAWKINS—1. Essential delivery room equipment.

SWEDISH COVENANT HOSPITAL

R. A. LIFVENDAHL, G. F. HIBBERT, G. L. ROSENE, and H. J. TIMERMAN—9. Vaginal hysterectomy; cesarean section case studies.

WASHINGTON BOULEVARD HOSPITAL

PAUL C. FOX—9. Operative and dry clinic.

WOMEN AND CHILDREN'S HOSPITAL

AMELIA GIRYOTAS, ELOISE PARSONS, PEARL STETLER, MARY EDITH WILLIAMS, and MAUDE HALL WINNETT—9. Surgical and gynecological operations.

BEATRICE TUCKER—9. Operations.

BERTHA VAN HOOSEN—9. Demonstration: Teaching gynecology by models.

Thursday

CHICAGO LYING-IN HOSPITAL

FRED L. ADAIR, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, H. CLOSE HESSELTINE, and FRANKLIN F. SNYDER—9. Operations.

FRED L. ADAIR and staff—2. Dry clinic:

WILLIAM J. DIECKMANN. Results of treatment of toxemia patients.

M. EDWARD DAVIS. Rational endocrine therapy in obstetrics and gynecology.

ALLAN T. KENYON. Metabolic influences of the steroid hormones.

MELBOURNE W. BOYNTON. Present-day management of the menopause.

KATSUJI KATO. Vitamin K deficiency in the newborn.

ALICE CHILDS. Heart disease in pregnancy.

DONALD M. SCHUITEMA. Subject to be announced.

COLUMBUS HOSPITAL

CHANNING W. BARRETT—9. Operations.

F. O. BOWE—9. Dry clinic.

COOK COUNTY HOSPITAL

E. W. FISCHMANN—9. Gynecological operations.

J. P. GREENHILL—10. Gynecological operations.

GRANT HOSPITAL

F. H. FALLS—9. Gynecological operation.

E. W. FISCHMANN—2. Gynecological operation.

HOLY CROSS HOSPITAL

P. E. LAWLER. Manikin demonstration.
F. F. FRAIDER and N. B. PAVLETIC. Hysterectomy.
F. J. SALETTA. Hysterectomy.

ILLINOIS MASONIC HOSPITAL

F. O. BOWE and PAULINE LANGE—9. Cesarean section.
DANIEL W. JEFFRIES and R. LEVISOHN—9. Ventral fixation of uterus.
HAROLD W. MILLER and GLEN NELSON—10. Hysterectomy.
ALLAN H. FERGUSON—10. Perineorrhaphy.
A. J. SCHOENBERG and M. OWEN WILKINS—11. Amputation of cervix.

NORWEGIAN-AMERICAN HOSPITAL

B. W. BREISTER—9. Indications for cesarean section.
P. F. SNYDER—11. Differential diagnosis of uterine tumors.

PASSAVANT MEMORIAL HOSPITAL

ARTHUR H. CURTIS and GEORGE H. GARDNER—9. Operative and dry clinic: Vaginal plastic procedures and abdominal total hysterectomies.
DAVID S. HILLIS and associates—2. Dry clinic: Cephalopelvic disproportion.

RESEARCH AND EDUCATIONAL HOSPITALS

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

ST. LUKE'S HOSPITAL

Staff—10. Operations.

Friday

CHICAGO LYING-IN HOSPITAL

FRED L. ADAIR, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, H. CLOSE HESSELTINE, and FRANKLIN F. SNYDER—9. Operations.
FRED L. ADAIR and staff—2. Dry clinic:
FRED L. ADAIR. Treatment of uterine prolapse.
FRANKLIN F. SNYDER. Subject to be announced.
H. CLOSE HESSELTINE. Management of chronic and resistant vaginal trichomoniasis.
HILGER P. JENKINS. Acute appendicitis of pregnancy.
LUCILE HAC. Transportation of gonococcal cultural material.
PRICILLA OUDA. Vomiting as a complication of obstetrical anesthesia.

COOK COUNTY HOSPITAL

WILLIAM H. BROWNE—9. Gynecological operations.
F. H. FALLS—10. Gynecological operations.
AUGUST DARO. Use of epinephrine in acute inversion of the uterus.
LOUIS RUDOLPH. Ring dystocia constriction.
JAMES E. FITZGERALD. Heart disease in pregnancy.
DAVID S. HILLIS. Recognition of cephalopelvic disproportion.
AUGUSTA WEBSTER. Vitamin K in pregnancy.

MICHAEL REESE HOSPITAL

WILLIAM RUBOVITS—9. Neugebauer-LeFort operation for prolapse.
RALPH A. REIS—9. Vaginal hysterectomy.
I. F. STEIN—9. Bilateral ovarian resection.
M. L. LEVENTHAL—9. Manchester operation for prolapse of uterus.

MT. SINAI HOSPITAL

A. E. KANTER and LOUIS RUDOLPH. Gynecological operations.
A. F. LASH. Vaginal hysterectomy.
HENRY BUXBAUM, CHARLES NEWBURGER, and A. H. GOLDFINE. Obstetrical clinic.

RESEARCH AND EDUCATIONAL HOSPITALS

F. H. FALLS and staff—2. Operative and dry clinic:
G. H. REZEK. Supracervical hysterectomy.
A. J. KOBAK. Report of three cases of interstitial ectopic pregnancy.
W. H. BROWNE. Vulvectomy.
R. A. LIFVENDAHL. LeFort operation for prolapse of uterus.
V. C. FREDA. Skin test for pregnancy.
F. H. FALLS. Vaginal hysterectomy under local anesthesia.

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

ST. ELIZABETH'S HOSPITAL

B. S. MILTON—9. Hysterectomy for fibroid tumors; discussion of pathology of fibroid tumors.
J. K. NARAT—1. Tumors of the uterus.
J. R. LAVIERI and F. J. WALSH—3. Surgical complications in obstetrics.

Days to be Announced

HOSPITAL OF ST. ANTHONY DE PADUA

M. A. WEISSKOPF. Obstetrical operations.

GENITO-URINARY SURGERY

Tuesday

EVANSTON HOSPITAL

JAMES I. FARRELL—9. Infections of the urinary tract.

MICHAEL REESE HOSPITAL

Staff—9. Dry clinic:
GUSTAV KOLISCHER. Carcinoma of breast successfully treated with macrophages.
IRVIN S. KOLL. Massive renal calculi.
HARRY C. ROLNICK. Radical perineal prostatectomy.
A. E. JONES. Hydronephrosis due to retroperitoneal lymphosarcoma.
IRVING SHAPIRO. Ureteral injuries in gynecological operations.
FREDERICK LIEBERTHAL. Tumors of kidney.
J. S. GROVE. Ureteral calculus in case of solitary kidney; urosepsis.

PRESBYTERIAN HOSPITAL

HERMAN L. KRETSCHMER, ROBERT HERBST, NORRIS J. HECKEL, and JAMES MERRICKS—9. Operative clinic and demonstration of cases.

RAVENSWOOD HOSPITAL

Staff—10. Demonstration of cases.

ST. JOSEPH HOSPITAL

Staff—9. Dry clinic.

WESLEY MEMORIAL HOSPITAL

VICTOR D. LESPINASSE—9. Operative and dry clinic.
DONALD K. HIBBS—10. Operative and dry clinic.

Wednesday

ALBERT MERRITT BILLINGS HOSPITAL

CHARLES B. HUGGINS—9. Operations.

HENROTIN HOSPITAL

C. O. MILLER—9. Neoplasm of the kidneys.

LORETTO HOSPITAL

CARL J. UTHOFF—9. Operative and dry clinic.

MERCY HOSPITAL

H. E. LANDES—9. Clinical cystometry, an aid in the diagnosis of disturbances of micturition.

MICHAEL REESE HOSPITAL

GUSTAV KOLISCHER, I. S. KOLL, J. S. EISENSTAEDT, H. C. ROLNICK, A. E. JONES, I. J. SHAPIRO, F. LIEBERTHAL, and J. S. GROVE—9. Operations.

MUNICIPAL TUBERCULOSIS SANITARIUM

D. F. RUDNICK—9. Genito-urinary tuberculosis.

ST. ANNE'S HOSPITAL

H. J. DOOLEY and C. C. SAELHOF—9. Operations.
H. J. DOOLEY—1. Anesthesia in genito-urinary surgery.
C. C. SAELHOF—1. Surgery of prostate in diabetics, discussion and demonstration.

ST. BERNARD'S HOSPITAL

A. J. SULLIVAN—9. Prostatic operations.

SWEDISH COVENANT HOSPITAL

B. E. FILLIS, J. T. GERNON, and K. D. KOHLSTEDT—9. Genito-urinary problems.

VETERANS ADMINISTRATION FACILITY

T. G. MCDOUGALL—9. Operations.

Thursday

ALEXIAN BROTHERS' HOSPITAL

JULIUS M. GLASSER—9. Prostatectomy, 2 cases.
EDWARD F. HESS—9. Dry clinic: Nephrotomy; cystoscopy and pyelography.
EDWARD W. WHITE—10. Prostatic electroresection; ureteral stone.
A. J. WOCHINSKI—10. Prostatic electroresection; fulguration of bladder tumor, 2 cases.

CHICAGO MEMORIAL HOSPITAL

VINCENT J. O'CONOR, JOHN P. O'NEIL, J. WILLIAM PARKER, and KENNETH SOKOL—9. Operative and dry clinic.

CHILDREN'S MEMORIAL HOSPITAL

HERMAN L. KRETSCHMER—9. Operative clinic and demonstrations: Urological problems in infancy and childhood.

COLUMBUS HOSPITAL

WILLIAM H. GEHL—9. Transurethral prostatic resection.

GRANT HOSPITAL

EDWARD F. HESS—9. Demonstration of cases.
WILLIAM H. GEHL—9. Operations.

ILLINOIS MASONIC HOSPITAL

WILLIAM H. GEHL and F. L. CHENOWETH—9. Nephrectomy.
C. OTIS RITCH and O. F. HARRIS—9. Suprapubic cystotomy.
EDWARD W. WHITE and CHARLES MURRAY—10. Prostatectomy.
R. B. GAINES—10. Nephrolithotomy.
CLARENCE C. SAELHOF—11. Cystoscopic examination.

MICHAEL REESE HOSPITAL

GUSTAV KOLISCHER, I. S. KOLL, J. S. EISENSTAEDT, H. C. ROLNICK, A. E. JONES, I. J. SHAPIRO, F. LIEBERTHAL, and J. S. GROVE—9. Operations.

NORWEGIAN-AMERICAN HOSPITAL

D. F. RUDNICK—10. Transurethral resection of prostate.

PASSAVANT MEMORIAL HOSPITAL

LEANDER W. RIBA—2. Dry clinic: Chronic female urethritis and its treatment.
L. L. VESEEN—3. Diagnosis and treatment of tumors of the testes.

ST. LUKE'S HOSPITAL

L. E. SCHMIDT and staff—9. Operative and dry clinic.

WASHINGTON BOULEVARD HOSPITAL

VINCENT J. O'CONOR—9. Operative and dry clinic.

Friday

MT. SINAI HOSPITAL

H. C. ROLNICK—9. Operations.

ST. ELIZABETH'S HOSPITAL

T. G. MCDOUGALL—3. Nephrectomy; discussion of tumors of the kidney.

Days to be Announced

COOK COUNTY HOSPITAL

HARRY CULVER, HARRY C. ROLNICK, D. F. RUDNICK, and L. L. VESEEN. Operations.

HOSPITAL OF ST. ANTHONY DE PADUA

OTTO JIRSA. Operative and dry clinic: Cystoscopy.

RESEARCH AND EDUCATIONAL HOSPITALS

C. M. MCKENNA, J. H. KIEFER and staff—9. Operative and dry clinic: Transurethral resection for hypertrophied prostate gland; suprapubic operation for hypertrophied prostate gland; nephrostomy and removal of aberrant vessels for hydronephrosis; repair of undescended testicle; total cystectomy following transplantation of ureters for bladder exstrophy; end-results of correction of hypospadias; end-results of repair of undescended testicle.
R. C. HERROLD—9. Evaluation of urinary antiseptics in treatment of infections.

ORTHOPEDIC SURGERY

Monday

CHILDREN'S MEMORIAL HOSPITAL

FREMONT A. CHANDLER, F. SEIDLER, C. N. PEASE, J. R. NORCROSS, and L. SMITH—2:30. Operative and dry clinic: Osteomyelitis; resection of ilium; rare infection of joint; tuberculous metatarsal and tuberculous ankle; patellar advancement; radial stripping for pronation deformity; unusual tumor of femur; unusual tumor of tibia; anterior dislocation of hip; torticollis, six cases; Erb's palsy; back knee; medial dislocation of patella; arachnodactylia, two cases; osteochondromatous of hip; overgrowth of spine.

RESEARCH AND EDUCATIONAL HOSPITALS

H. B. THOMAS, F. W. HARK, C. N. LAMBERT, and S. L. ODGERS—2. Operative and dry clinic: Fusion of tuberculous hip; end-results of tuberculosis of hip and spine; tendon transplant to shoulder; results of tendon transplant for brachial plexus injury to shoulder; use of shift casts in treatment of scoliosis.

Tuesday

CHILDREN'S MEMORIAL HOSPITAL

FREMONT A. CHANDLER, F. SEIDLER, C. N. PEASE, J. R. NORCROSS, and L. SMITH—9. Operative clinic: Extraperitoneal resection of obturator nerve, two cases; Lowman operation for flat feet; triple arthodesis; Chandler patellar advancement operation; spine fusion; torticollis, four to five weeks of age; hip fusion operation; popliteal neurectomy.

COOK COUNTY HOSPITAL

ARTHUR H. CONLEY and DONALD MILLER—9. Operations.
MARCUS H. HOBART and FELIX JANSEY—10. Operative clinic: Shoulder injuries.

EVANSTON HOSPITAL

R. C. LONERGAN—9. Operative clinic: Internal derangement of knee.

MICHAEL REESE HOSPITAL

PHILIP LEWIN, D. H. LEVINTHAL, SIDNEY SIDEMAN, IRVING WOLIN, C. N. PEASE, FRANK GLASSMAN, and J. G. FINDER—9. Operative clinic: Spinal fusion for scoliosis; Hoke arthrodesis for paralytic foot; laminectomy for intervertebral disc protrusion; poliomyelitis reconstruction; knee joint operation; hip joint operation.

ST. JOSEPH HOSPITAL

Staff—9. Demonstrations: Lantern slides and moving pictures of fractures.

SHRINERS' HOSPITAL FOR CRIPPLED CHILDREN

B. H. MOORE and H. A. SOFIELD—9. Operative clinic.

Wednesday

COOK COUNTY HOSPITAL

PHILIP LEWIN and SIDNEY SIDEMAN—9. Operative clinic.
DANIEL H. LEVINTHAL and staff—9. Dry clinic.
DANIEL H. LEVINTHAL—2. Operative clinic: Derangement of the knee joint; spinal fusion; arthroplasty of hip using vitallium cup; arthrodesis of foot for poliomyelitis.

LORETTO HOSPITAL

BEVERIDGE H. MOORE, JAMES A. VALENTINE, and FRED CARLS—10. Operative and dry clinic.

MICHAEL REESE HOSPITAL

Staff—2. Dry clinic: Dunlop traction treatment of transcondylar fractures of the elbow in children; Risser method in scoliosis; club feet; Legg-Calve-Perthes lesion; tuberculosis of the hip joint; tuberculosis of the spine; chronic osteomyelitis; ricketic deformities; postoperative laminectomy for disc protrusion; open reduction of congenital hip dislocation; flexorplasty of thumb; spina bifida with paraplegia; vitallium cup hip arthroplasty; leg equalization by epiphyseal arrests; leg shortening.

PASSAVANT MEMORIAL HOSPITAL

PAUL B. MAGNUSON and JAMES K. STACK—3. Dry clinic: Joint debridement; cases illustrating end-results of treatment of traumatic arthritis.

ST. ANNE'S HOSPITAL

J. J. CALLAHAN and R. E. MEANY—9. Operations.

ST. LUKE'S HOSPITAL

HENRY B. THOMAS—10. Bone changes in connection with thyroid disease.
FRED W. HARK—10. Bone changes in connection with sickle-cell anemia.
CLAUDE N. LAMBERT—10. Bone tumors.
H. E. MOCK, ALVIN R. MORROW, CHARLES E. SHANNON, JOHN L. LINDQUIST, and DONNELL C. HOWE—11. Dry clinic: Conservative treatment of fractures of neck of femur; fractures of fingers and toes; supracondylar fractures of humerus; malunited fractures of ankle.
JOHN W. ELLIS—11. Compression fractures of vertebrae using Ryerson hyperextension device.
EDWIN W. RYERSON—1:30. Dry clinic: Shelf operation at hip; reconstruction of hip joint; osteotomy to produce back-knee.
FREMONT A. CHANDLER—2. Operative clinic: Aseptic necrosis of head of femur.
ROBERT E. RITTER—2:30. Arthroplasty.
HAROLD A. SOFIELD—3. Discussion of leg lengthening operations; indications and technique.
Open forum—3:30.

SHRINERS' HOSPITAL FOR CRIPPLED CHILDREN

Staff—3. Clubfoot clinic.

VETERANS ADMINISTRATION FACILITY

S. K. LIVINGSTON—10. Dry clinic: Peripheral vascular diseases.

Thursday

ALEXIAN BROTHERS' HOSPITAL

GEORGE L. APPELBACH—10. Demonstration of examination for lower back pain; non-union of neck of femur.

ALBERT MERRITT BILLINGS HOSPITAL

DALLAS B. PHEMISTER and C. HOWARD HATCHER—9. Surgery of the hip joint; growth arrest operations.

COLUMBUS HOSPITAL

FREDERICK MUELLER—9:30. Dry clinic.

COOK COUNTY HOSPITAL

FRANK G. MURPHY and WALTER FISCHER—9. Operations.
E. J. BERKHEISER—10. Operations.
ARTHUR H. CONLEY and staff—10. Dry clinic.

GRANT HOSPITAL

JOSEPH E. ALLEGRETTI—2. Treatment of arthritis.
GEORGE L. APFELBACH—2. Demonstration of examination for lower back pain; open reduction of fracture.

HOLY CROSS HOSPITAL

C. P. GALANTI. Rare bone tumors.

ILLINOIS MASONIC HOSPITAL

CHARLES N. PEASE—9. Fixation of hip.
WALTER R. FISCHER—10. Osteomyelitis.

MICHAEL REESE HOSPITAL

PHILIP LEWIN, D. H. LEVINTHAL, SIDNEY SIDEMAN, IRVING WOLIN, C. N. PEASE, FRANK GLASSMAN, and J. G. FINDER—9. Operative clinic: Spinal fusion for scoliosis; Hoke arthrodesis for paralytic foot; laminectomy for intervertebral disc protrusion; poliomyelitis reconstruction; knee joint operation; hip joint operation.

SHRINERS' HOSPITAL FOR CRIPPLED CHILDREN

B. H. MOORE and H. A. SOFIELD—9. Operative clinic.

RESEARCH AND EDUCATIONAL HOSPITALS

H. B. THOMAS, F. W. HARK, C. N. LAMBERT, and S. L. ODGERS—2. Operative and dry clinic: Open reduction for slipped epiphysis of the femur and insertion of fibular graft through neck; end-results of hip joint surgery, particularly fractures and epiphyseal separations.

Friday

COOK COUNTY HOSPITAL

FRED W. HARK and CLAUDE N. LAMBERT—9. Operations.
H. KELIKIAN and GRAHAM A. KERNWEIN—10. Operations.

MT. SINAI HOSPITAL

C. L. JACOBS and LEO F. MILLER—9. Operations.

PASSAVANT MEMORIAL HOSPITAL

EMIL HAUSER—10. Case presentations: Fusion of hip for tuberculosis; spinal fusion, presenting an unusual complication; Brodie's abscess, illustrating results of chemotherapy; after-result of treatment of delayed union in fracture of humerus; after-treatment for fracture of calcaneum.

ST. MARY OF NAZARETH HOSPITAL

L. M. CZAJA—9. Operations.

FRACTURES AND OTHER TRAUMAS

Monday

COOK COUNTY HOSPITAL

WILLIAM R. CUBBINS, J. J. CALLAHAN and C. S. SCUDERI—1. Operative clinic.

Tuesday

CHICAGO MEMORIAL HOSPITAL

T. C. BROWNING, EDWARD L. COMPERE, ARTHUR H. CONLEY, EMILE C. DUVAL, C. R. G. FORRESTER, A. H. MASON, FRED M. MILLER, S. PERRY ROGERS, HORACE STIMSON, and GEORGE M. LANDAU—9. Operative and dry clinic:

C. R. G. FORRESTER. Shoulder fractures.
A. H. MASON. Fractures of the scaphoid.
FRED M. MILLER and T. C. BROWNING. Fractures of foot and ankle.
EDWARD L. COMPERE. Vitamin D and calcium in fracture repair.
S. PERRY ROGERS. Vitallium in fractures.
EMILE C. DUVAL. Internal semilunar cartilage as a factor in industrial surgery.
HORACE STIMSON. Treatment of fractures of spine.

EVANSTON HOSPITAL

MARCUS H. HOBART. Malunited Colles' fracture.
DWIGHT F. CLARK. Intramedullary splinting with cow's horn; four-year end-result.

RAVENSWOOD HOSPITAL

Staff—10. Demonstration of cases.

ST. JOSEPH HOSPITAL

HUGH MCKENNA—9. Demonstrations; Motion pictures and slides of fractures of the neck of femur and demonstration of a device used in securing x-ray films during the stage of reduction.

WASHINGTON BOULEVARD HOSPITAL

ARTHUR R. METZ—9. Operative and dry clinic.

Wednesday

CHILDREN'S MEMORIAL HOSPITAL

ALBERT H. MONTGOMERY, JAY IRELAND, and W. J. POTTS—2:30. Demonstration of cases: Fractures of forearm; supracondylar fractures of humerus; bone cysts; skull fractures.

COOK COUNTY HOSPITAL

GEORGE L. APFELBACH—2. Operative clinic: Flange operation for fractured neck of femur; open reduction of the forearm with intramedullary peg; reconstruction of malunited Colles' fracture.

HENROTIN HOSPITAL

RALPH A. KORDENAT—9. Operations.
JOHN A. GRAHAM—9. Demonstration, with lantern slides: Fracture of distal end of radius.
JOHN J. EICHSTADT—9. Dry clinic: Care of fractures.

JACKSON PARK HOSPITAL

F. G. MURPHY—9. Operations.

MERCY HOSPITAL

F. C. JACOBSEN—2. Industrial fractures.
JOHN D. CLARIDGE—2. Fractures of the humerus.
J. M. LEONARD—4. Forearm fractures.

PASSAVANT MEMORIAL HOSPITAL

PAUL B. MAGNUSON and JAMES K. STACK—3. Dry clinic; Results of treatment of various types of fractures of the neck of the femur, treated by selected methods for the individual case. The effect of trauma on prolongation of the symptoms of arthritis. Arthritis resulting purely from trauma, with demonstration of pathological sections and specimens in color.

ST. ANNE'S HOSPITAL

J. J. CALLAHAN—1. Dry clinic: Shoulder injuries; congenital dislocation of patella.
T. E. MEANY—1. Dry clinic: Fracture of femur in children —Paget's disease.

SWEDISH COVENANT HOSPITAL

O. T. ROBERG and O. T. ROBERG, JR.—9. Fractures.

Thursday

COLUMBUS HOSPITAL

GEORGE N. BEECHER—9. Dry clinic.

COOK COUNTY HOSPITAL

WILLIAM R. CUBBINS and JAMES J. CALLAHAN—1. Operations.

NORWEGIAN-AMERICAN HOSPITAL

H. A. SOFIELD—11. Steel pin fixation of fractures of neck of the femur.

Friday

COOK COUNTY HOSPITAL

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

Staff—1. Demonstration on cadaver: Fractures and traumatic surgery.

PRESBYTERIAN HOSPITAL

Staff—9. Operative and dry clinic:
EDWIN M. MILLER. Improved methods in dealing with supracondylar fractures of the elbow; resection of tumor of metacarpal bone with free graft from tibia; x-rays, microscopic pathology and clinical result.
W. J. POTTS. Improved method of immobilization of fractures of both bones of leg.
EGBERT H. FELL. Fractures of the capitellum of the humerus.
EDWARD L. COMPERE. Evaluation of methods of internal fixation of fractures of neck of the femur.
E. J. BERKHEISER. Dislocations of cervical vertebrae; arthrodesis of shoulder; tendon transplants.
KELLOGG SPEED. Reconstruction problems following trauma in hip region; illustrated talk on wounds of war; fractures about elbow.

Days to be Announced

HOSPITAL OF ST. ANTHONY DE PADUA

F. W. SLOBE. Operative clinic: Fractures; tendon sutures; care of wounds; treatment of burns.

WESLEY MEMORIAL HOSPITAL

Staff—9. Operative and dry clinic:
O. H. HORRALL. Fractures of the os calcis, colored motion pictures. Operation for semilunar cartilage; open reduction of fracture of the tibia.
F. M. JANSEY. Fractures of the pelvis. Operation for open reduction of fracture of the humerus.
H. KELIKAN. Fractures involving the knee joint. Operation for open reduction, fracture of the shaft of the femur.
P. H. KREUSCHER. Fractures about the hip joint. Colles and Pott's fractures. Operation for recurrent dislocation of the shoulder; removal of semilunar cartilage.
P. B. MAGNUSON. Fractures and other injuries of the spine.
RALPH MACDONALD. Fractures about the shoulder.
K. PENHALE. Traumatic injuries of the face and jaws.

THORACIC SURGERY

Tuesday

COOK COUNTY HOSPITAL

JEROME R. HEAD—10. Operative clinic: Lung abscess, empyema, and other intrathoracic infections.
R. B. BETTMAN—2. Operations: Intrathoracic tumors.

EVANSTON HOSPITAL

JEROME R. HEAD—9. Operations.

RESEARCH AND EDUCATIONAL HOSPITALS

WILLARD VAN HAZEL—9. Operative and dry clinic: Thoracoplasty; thoracotomy for chronic empyema; discussion of surgical drainage of lung abscess; lobectomy for bronchiectasis.

Wednesday

ST. LUKE'S HOSPITAL

WILLARD VAN HAZEL—9. Operative and dry clinic: Thoracoplasty. Treatment of lung abscess.

VETERANS ADMINISTRATION FACILITY

JEROME R. HEAD—11. Operative and dry clinic: Stages I and II of a new muscle splitting thoracoplasty; extrapleural pneumothorax; Monaldi suction treatment of tuberculous cavities.

Thursday

ALBERT MERRITT BILLINGS HOSPITAL

W. E. ADAMS—9. Intrathoracic operations with special reference to reduced lung function and partial pressure anesthesia.

ALEXIAN BROTHERS' HOSPITAL

MINAS JOANNIDES and LOUIS J. MILLER—9. Operative clinic: First-stage thoracoplasty; extrapleural pneumolysis, axillary approach; lobectomy for bronchiectasis. Dry clinic: Hedblom's syndrome, clinical and radiological aspects; chronic suppuration of the lung in relation to carcinoma of the lung.

ILLINOIS MASONIC HOSPITAL

MINAS JOANNIDES and W. E. KEESEY—9. Operative clinic.
MINAS JOANNIDES and LOUIS J. MILLER—9. Monodal drainage, extrapleural pneumolysis, phrenic neurectomy and anterior thoracoplasty.
LOUIS J. MILLER—9. Surgical aspects of pulmonary abscess and primary carcinoma of the lung.

MICHAEL REESE HOSPITAL

S. L. GOLDBERG—2. Stab wound of pericardium and lung.
NATHAN N. CROHN—2. Concepts in treatment of empyema.
MAX BIESENTHAL—2. A ten-year review of collapse therapy in treatment of tuberculosis.
R. B. BETTMAN and WILLIAM TANNENBAUM—2. Surgical approach to thoracic esophagus.
R. B. BETTMAN—2. Interesting cases in thoracic surgery.
B. B. LENNON—2. Anesthesia in thoracic surgery.

Friday

ALBERT MERRITT BILLINGS HOSPITAL

W. E. ADAMS—9. Thoracoplasty; lobectomy.

MICHAEL REESE HOSPITAL

R. B. BETTMAN and W. TANNENBAUM—9. Operations.

MUNICIPAL TUBERCULOSIS SANITARIUM

MINAS JOANNIDES—9. Collapse therapy clinic.

PRESBYTERIAN HOSPITAL

JOHN M. DORSEY—9. Operative and dry clinic: Intrathoracic tumors and diaphragmatic hernia.

NEUROSURGERY

Tuesday

ALBERT MERRITT BILLINGS HOSPITAL

PAUL BUCY—9. Operations.

RESEARCH AND EDUCATIONAL HOSPITALS

ERIC OLDBERG, PERCIVAL BAILEY, W. A. GUSTAFSON, and MILTON TINSLEY—9. Operations and demonstrations.

ST. LUKE'S HOSPITAL

ERIC OLDBERG and WESLEY A. GUSTAFSON—9. Operations and demonstrations.

Wednesday

COOK COUNTY HOSPITAL

HAROLD C. VORIS—9. Dry clinic: Operative treatment of head injuries.
ADRIEN VERBRUGGHEN and associates—1. Operations.

PASSAVANT MEMORIAL HOSPITAL

LOYAL DAVIS and JOHN MARTIN—2. Dry clinic: Discussion of results of surgical treatment of essential hypertension; discussion of tumors of the nasopharynx, producing intracranial symptoms.

RESEARCH AND EDUCATIONAL HOSPITALS

ERIC OLDBERG, PERCIVAL BAILEY, W. A. GUSTAFSON, and MILTON TINSLEY—9. Operations and demonstrations.

Thursday

ALBERT MERRITT BILLINGS HOSPITAL

PAUL BUCY—9. Operations.

COOK COUNTY HOSPITAL

ADRIEN VERBRUGGHEN and associates—10. Dry clinic: Concussion of the brain.

MERCY HOSPITAL

HAROLD C. VORIS—9. Protruding intervertebral disc.

MICHAEL REESE HOSPITAL

ADRIEN VERBRUGGHEN—2. Operative and dry clinic.

RESEARCH AND EDUCATIONAL HOSPITALS

ERIC OLDBERG, PERCIVAL BAILEY, W. A. GUSTAFSON, and MILTON TINSLEY—9. Operations and demonstrations.

Friday

PRESBYTERIAN HOSPITAL

ADRIEN VERBRUGGHEN—9. Operations.

RESEARCH AND EDUCATIONAL HOSPITALS

ERIC OLDBERG, PERCIVAL BAILEY, WESLEY A. GUSTAFSON, and MILTON TINSLEY—9. Operations and demonstrations.

ST. LUKE'S HOSPITAL

ERIC OLDBERG and WESLEY A. GUSTAFSON—9. Operations and demonstrations.

OPHTHALMOLOGY

Tuesday

CHICAGO MEMORIAL HOSPITAL

FRANCIS M. CRAGE, HERMAN P. DAVIDSON, and GLENWAY NETHERCUT—2. Operative and dry clinic.

EVANGELICAL HOSPITAL

G. HENRY MUNDT, J. R. SMITH, EVAN A. MILLER, and J. H. F. O'NEIL—9. Operations.

EVANSTON HOSPITAL

G. R. SOPER, GLENN J. GREENWOOD, and R. H. HENDERSON—9. Operations.

GRANT HOSPITAL

OSCAR H. KRAFT—9. Operative and dry clinic.

ILLINOIS EYE AND EAR INFIRMARY

THOMAS D. ALLEN, V. M. LEECH, and G. W. NETHERCUT—2. Operative clinic: Cataracts; ptosis; glaucoma.

PASSAVANT MEMORIAL HOSPITAL

SANFORD GIFFORD and associates—9. Dry clinic: Retina detachment; retinoplasty; plastic surgery of the eyelids.

RESEARCH AND EDUCATIONAL HOSPITALS

H. J. SMITH—9. Management of foreign bodies.
HALLARD BEARD—9. Surgical treatment of glaucoma.

ST. JOSEPH HOSPITAL

Staff—9. Operative and dry clinic.

ST. LUKE'S HOSPITAL

ELMER VORISEK—2. Demonstration of cases.

WESLEY MEMORIAL HOSPITAL

T. P. O'CONNOR—9. Operative and dry clinic.
WILLIAM A. MANN and IRVING PUNTENNEY—10. Operative and dry clinic.

Wednesday

CHILDREN'S MEMORIAL HOSPITAL

RICHARD GAMBLE and associates—4. Dry clinic.

COOK COUNTY HOSPITAL

JAMES E. LEBENSOHN and WILLIAM F. MONCREIFF—9. Operations.

ILLINOIS EYE AND EAR INFIRMARY

PETER C. KRONFELD—9. Gonioscopy.
KATHARINE CHAPMAN—9. Orthoptic clinic.
ROBERT VON DER HEYDT—3. Slit lamp demonstration.

MERCY HOSPITAL

CARL F. SCHAUB and L. G. HOFFMAN—9. Operative and dry clinic. Demonstration of verified cases of opticochiasmatic arachnoiditis.

ST. BERNARD'S HOSPITAL

C. P. SULLIVAN. Cataract operations.

SWEDISH COVENANT HOSPITAL

R. A. DAVIS—9. Operative and dry clinic: Glaucoma.

Thursday

COLUMBUS HOSPITAL

MICHAEL GOLDENBURG—9. Operations.

EVANGELICAL HOSPITAL

G. HENRY MUNDT, J. R. SMITH, EVAN A. MILLER, and J. H. F. O'NEIL—9. Operations.

ILLINOIS EYE AND EAR INFIRMARY

LOUIS G. HOFFMAN—9. Operative and dry clinic· Verhoeff suture.
E. K. FINDLAY—10. Dry clinic.
E. K. FINDLAY and DR. CORBOY—2. Operative clinic.

ST. LUKE'S HOSPITAL

E. V. L. BROWN—9. Demonstration of cases.
FRANK BRAWLEY and JAMES W. CLARK—2. Demonstration of cases.

Friday

COOK COUNTY HOSPITAL

SANFORD R. GIFFORD—9. Operations.
EDWARD A. ROLING—10. Operations.

ILLINOIS EYE AND EAR INFIRMARY

PETER C. KRONFELD—9. Gonioscopy.
KATHARINE CHAPMAN—9. Orthoptics.
SAMUEL J. MEYER—2. Operative clinic: Cataracts; glaucoma.

RESEARCH AND EDUCATIONAL HOSPITALS

CARL APPLE—9. Recession operation for strabismus.
J. W. CLARK—9. Intracapsular extraction of cataract.

ST. ELIZABETH'S HOSPITAL

F. A. DULAK and OSCAR CLEFF—3. Operative and dry clinic: Glaucoma.

ST. LUKE'S HOSPITAL

RICHARD GAMBLE—3. Demonstration of cases.

OTOLARYNGOLOGY

Monday

ILLINOIS EYE AND EAR INFIRMARY

SAMUEL SALINGER—2. Operative and dry clinic: Plastic surgery about the nose.

Tuesday

ALBERT MERRITT BILLINGS HOSPITAL

JOHN R. LINDSAY—2. Exhibit and demonstration: Suppurations of the petrous pyramid; anatomical, pathological, and roentgenological considerations.

CHICAGO MEMORIAL HOSPITAL

OSCAR CLEFF, ALFRED LEWY, ROBERT LEWY, JAMES B. MCBEAN, GORDON H. SCOTT, and RICHARD W. WATKINS—2. Operations.

COOK COUNTY HOSPITAL

JACOB LIFSCHUTZ—2. Operative and dry clinic.
RAYMOND W. KERWIN—2. Operative and dry clinic.

EVANGELICAL HOSPITAL

G. HENRY MUNDT, J. R. SMITH, EVAN A. MILLER, and J. H. F. O'NEIL—9. Operations.

EVANSTON HOSPITAL

T. C. GALLOWAY, H. C. BALLENGER, and L. J. LAWSON. Operations.
T. C. GALLOWAY. Anerobic bone infections about the head.

ILLINOIS EYE AND EAR INFIRMARY

T. P. O'CONNOR—9. Plastic surgery, demonstration of patients, lantern slides.
CARL CRISTOPH—9. Operative clinic: Radical mastoid.
E. J. BLONDER—2. Labyrinthine diagnosis and demonstration of the galvanic following reaction.
M. A. GLATT—2. Operative clinic: Radical mastoid and external frontal.

RESEARCH AND EDUCATIONAL HOSPITALS

P. H. HOLINGER and A. H. ANDREWS, JR.—9. Bronchoscopies.

F. L. LEDERER and staff—2. Otorhinolaryngological operations.

ST. JOSEPH HOSPITAL

Staff—9. Operative and dry clinic.

WESLEY MEMORIAL HOSPITAL

T. P. O'CONNOR and LEONARD C. DELOZIER—10. Operative and dry clinic.
TAI TONG CHING—11. Operative and dry clinic.

Wednesday

COOK COUNTY HOSPITAL

S. J. PEARLMAN—10. Operative and dry clinic.
JACOB LIFSCHUTZ—2. Operative and dry clinic.

ILLINOIS EYE AND EAR INFIRMARY

DR. WOODRUFF. Laryngeal sinus and ear cases; endoral, mastoid and sinus surgery.
T. P. O'CONNOR—2. Operative clinic.

JACKSON PARK HOSPITAL

NOAH FOX—9. Operations.

PASSAVANT MEMORIAL HOSPITAL

JOHN F. DELPH and associates—9. Dry clinic: Laryngoscopy, bronchoscopy, esophagoscopy; presentation of exhibits and cases; carcinoma of larynx.

ST. ANNE'S HOSPITAL

J. W. HAYDEN—9. Operations.

ST. MARY OF NAZARETH HOSPITAL

F. J. PISZKIEWICS—9. Otological operations.

VETERANS ADMINISTRATION FACILITY

MAX CUTLER—11. Dry clinic: Carcinoma of the mouth and larynx. Problems in diagnosis and treatment; indications for surgery and irradiation in laryngeal carcinoma; presentation of cases.

Thursday

ALBERT MERRITT BILLINGS HOSPITAL

JOHN R. LINDSAY—2. Exhibit and demonstration: Suppurations of the petrous pyramid; anatomical, pathological, and roentgenological considerations.

COOK COUNTY HOSPITAL

RAYMOND W. KERWIN—2. Operative and dry clinic.

EVANGELICAL HOSPITAL

G. HENRY MUNDT, J. R. SMITH, EVAN A. MILLER, and J. H. F. O'NEIL—9. Operations.

GRANT HOSPITAL

CARROLL W. STUART—2. Oral surgery.
S. H. SOBOROFF—2. Nose and throat operation.

ILLINOIS EYE AND EAR INFIRMARY

E. J. BLONDER—9. Operative and dry clinic: Radical mastoid.
M. A. GLATT—2. Osteomyelitis of the frontal bone.

ILLINOIS MASONIC HOSPITAL

ALVA SOWERS—9. Tonsillectomy.
ARTHUR H. GEIGER—9. Deviated septum.
MAURICE H. COTTLE and MILTON LEVISOHN—10. Mastoidectomy.
HERBERT E. TAYLOR—11. Drainage maxillary sinus.
JOHN GILMORE and PERRY J. MELNICK—12. Diagnosis and treatment of carcinoma.

MERCY HOSPITAL

PETER P. LEDERMANN—9. Disturbances of equilibrium.
CARL H. CHRISTOPH—9. Laryngeal malignancies.
HERBERT NASH—9. Endaural operations.
G. T. JORDAN—9. Anatomy of the temporal bone.

MUNICIPAL TUBERCULOSIS SANITARIUM

GEORGE HOLMES—2. Bronchoscopy in tuberculosis.

NORWEGIAN-AMERICAN HOSPITAL

M. A. GLATT—9. Control of hemorrhage in tonsillectomy.
J. W. HARNED—9:30. Danger signals in mastoid infection.

ST. LUKE'S HOSPITAL

PAUL H. HOLINGER and ALBERT H. ANDREWS, JR.—2. Peroral endoscopy; clinical presentation of bronchial and esophageal cases.
HORACE R. LYONS, WALTER H. THEOBALD and staff—3. Operative and dry clinic: Sulfanilamide therapy in acute mastoiditis and acute otitis media; Proetz treatment for sinusitis.

Friday

CHILDREN'S MEMORIAL HOSPITAL

PAUL H. HOLINGER and ALBERT H. ANDREWS—10:30. Bronchoscopic clinic.

COOK COUNTY HOSPITAL

JACOB LIFSCHUTZ, T. C. GALLOWAY, and R. W. KERWIN—10. Operative and dry clinic.

ILLINOIS EYE AND EAR INFIRMARY

ALFRED LEWY—2. Operative clinic.

MT. SINAI HOSPITAL

S. M. MORWITZ—9. Operations.

RESEARCH AND EDUCATIONAL HOSPITALS

L. W. SCHULTZ—9. Operative and dry clinic: Cleft palate operation; presentation of interesting cases in oral surgery; stabilization of joints by injecting sclerosing agent; bilateral cleft lip operation; bilateral temporomandibular dislocation for 2½ months, reduction, end-results with motion pictures; vitalium cup arthroplasty of temporomandibular joint, presentation of case.

ST. ELIZABETH'S HOSPITAL

J. G. WEBER and T. V. CARELLI—3. Operative and dry clinic: Complications in mastoid surgery.